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Original Articles and Clinical Cases.

HUGHLINGS JACKSON AS PIONEER IN NERVOUS PHYSIOLOGY AND PATHOLOGY.

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THERE is no honour of which I could be more sensible than that of being called upon by the Neurological Society to deliver the Hughlings Jackson Lecture; no task which I should undertake with greater pleasure than that of reviving my recollection of the nervous studies, which, in the early period of my professional life, constituted my greatest interest; no opportunity I could more cordially welcome than that of paying my homage of affection and respect to my old friend and fellow worker.

The interpretation which I put upon the duty entrusted to me by the Society is, that I should endeavour to set forth the principles in neurology established by Hughlings Jackson, and the reasons why, in this country, he is the acknowledged chief among neurologists.

This may seem to be but a humble task which I have set myself, but if I can get to the core of his teaching I do not think that I can render a greater service to neurology, whether as a department of practical medicine or as a branch of biological science, or that I can do better for the

younger generation of neurologists than to send them back to the study of the original writings, by which clear and definite ideas of nervous phenomena and nervous action were reached. We accept placidly the results of the labours of predecessors, but it is stimulating and inspiring to follow the steps by which order has been evolved from confusion, and to see pioneers wrestling with difficulties.

Hughlings Jackson has given to the world no such monumental treatise on diseases of the nervous system as that of Gowers.

We do not owe to him any such superb series of clinical and pathological descriptions and investigations as those which have made the name of Charcot illustrious.

He has not engaged in the experimental investigations into the functions of the different parts of the nervous system, which form the basis of nervous physiology, and from Bell onwards, in the hands of Claude Bernard, Majendie, Flourens, Brown-Sequard, Fritz and Hitzig, and Ferrier, to Horsley, Schäfer, Sherrington, Mott, and many others, have enabled us to understand the mechanism of the brain and cord.

He has taken no part in the microscopic unravelling of the intricate and complicated arrangements of cells and fibres, by means of which the nerve centres exercise their marvellous functions.

This makes it all the more extraordinary that, as is the case, we accord to him unanimously the first place among those who have contributed to neurology as a science.

The great reason is that he has been a pioneer in nervous physiology and pathology both in methods and results, has continuously raised our ideas of nervous action to a higher plane of thought, and has penetrated more deeply than even experimental science into the relations of the different nerve centres with each other and with the periphery, bringing to bear upon nervous physiology and pathology the speculations of evolutionary philosophy.

There are two kinds of knowledge, one consisting in the accumulation and verification of facts and their arrangement in their natural relation; the other of a more pro-

found character, comprehending the underlying significance of phenomena. It is this last to which Hughlings Jackson has so largely contributed.

The keynote to his teachings may be given in his own words. "The same fundamental principles apply to all nervous diseases whatsoever, from such as paralysis of an external rectus up to insanity."

All his writings are developments of this thesis.

Hughlings Jackson was the first in point of time to break through the barriers which experiments on animals had erected between the cerebrum and the lower nerve centres.

The motor or kinæsthetic functions of the cerebral cortex are now so familiar that we can scarcely imagine that any question could be entertained with regard to them, but when Hughlings Jackson entered upon the study of nervous diseases he was faced by the experimental facts that when the hemispheres were sliced away there was no evidence of loss of any particular faculty, but a gradual diminution of volition, that the hemispheres could be removed altogether, apparently leaving the motor apparatus of the nervous system intact, that the headless frog turned over when laid on its back, jumped, and rubbed vinegar off its back, that the pigeon with its cerebral hemispheres removed, while inert and impassive, walked when pushed and flew when thrown in the air, that the rabbit, deprived of its brain, started at a noise, screamed when pinched, hopped in a natural manner when pushed. The conclusion seemed inevitable that the brain proper had nothing to do with movements.

Hughlings Jackson anticipated the experimental demonstrations of Hitzig and Ferrier and showed that the experiments made by disease could be used in the interpretation of the anatomy and physiology of the nervous system, and that movements could be induced by the excitation of portions of the cerebral cortex, the removal of which did not give rise to paralysis.

The two great subjects with which his name will always be associated are the study of convulsions, with the elucida-

tion of epilepsy to which this study led, and the theory of evolution and dissolution as applied to the nervous system.

When he first gave his attention to the question of convulsions no definite ideas were entertained as to their causation, and it would be extremely interesting, were there time, to follow the stages by which he reached the conclusions with which we are now familiar.

He has informed us that the origin of his special investigations on epilepsy was a case in which right unilateral convulsion was followed by temporary defect of speech, and we find him saying, in the comments on a case of this kind reported in the *Medical Times and Gazette* as early as August, 1864, that the brain substance must be temporarily ill-nourished by inadequate blood supply, just as it would be permanently damaged by plugging of the arterial trunk. In the first we have unilateral convulsion, in the second, unilateral paralysis, hemiplegic epilepsy, and epileptic hemiplegia respectively. Convulsion, he continues, is closely allied to paralysis—paralysis often follows convulsion and convulsion often begins in parts already paralysed.

Chorea also, especially hemichorea, was shown to teach the same lesson, that irregular movements and paralysis of a given set of muscles indicate lesion of different degree in the same nerve centre.

In April, 1866 (*Medical Times and Gazette*), he already considered the term epilepsy to imply not simply liability to convulsive attacks, but to include sudden and temporary loss of function of any nerve centre, loss of sight, of consciousness, of speech, spasm of hand, face, or leg, and to have as its cause an affection of the nervous tissue in a particular arterial region, and in 1867 (*Lancet*, "Regional Palsy and Spasm"), while speaking of genuine epilepsy as an insoluble problem, he proposed to begin the investigation of it with a simpler form of convulsion and to try and find out what condition of the nervous matter it was in a particular part, the corpus striatum, which permitted occasional spasm of the muscles which this part empowers, and to study phenomena so as to get to know the portions of the nervous centres, affection of which was attended with sudden (1) loss

of speech for a while, (2) coloured vision, (3) spasm of arm, (4) smell, (5) transient loss of consciousness, (6) sudden fury, (7) convulsions.

Auræ were the first outward signs of the first local changes inside, and just as unilateral palsy and spasm were contrasted he would contrast these. As in hemiplegia we endeavour to ascertain the nature of the local lesion which has given rise to the paralysis, so we should inquire into the nature of the morbid change giving rise to convulsion.

In 1868, in a paper on the physiology of language (*Medical Times and Gazette*) is the first mention of evolution which I have met with.

Even the work of an ordinary kind which Hughlings Jackson has done in connection with diseases of the nervous system has not been done in an ordinary way, and his records of cases are illumined by flashes of insight which make the perusal of them interesting and profitable. Instances are his early recognition of syphilis as a cause of disease in the nerve centres through gummata in the pia, and through vascular lesions, the remarkable collection of cases illustrating localising lesions in the bulb, pons, and crura cerebri, the examples of unilateral convulsions of cortical origin, the illustrations of the association of right hemiplegia with loss or defect of speech. In 1864 he had already seen seventy cases of loss or defect of speech, in all except one of which there was hemiplegia on the right side, and had arrived independently at the conclusion that the left hemisphere was specially concerned in speech, all but anticipating and certainly paving the way for the immediate acceptance of Broca's localisation of the downward starting point for words. Mention also must be made of his early and persistent advocacy of the routine use of the ophthalmoscope and of his discussion of the significance of optic neuritis and its relation to intracranial disease.

It is scarcely work of an ordinary kind which is found in his lecture on the study of diseases of the nervous system in the *London Hospital Reports* for 1864.[1] In it he inculcates the practice which he himself always followed, and which in his hands has led to the elucidation of so

[1] *Vide* post, p. 367.

many nervous problems, that the observation of nervous symptoms must not only be *minute* but *precise*, and lays down the rule, now familiar to everyone, that we must pursue three distinct lines of thought in investigating nervous diseases with regard to :—(1) Disease of tissue ; (2) damage to organ ; (3) disorder of function ; or, as he puts it elsewhere, consider separately their pathology, anatomy and physiology.

From the symptoms, which are physiological, we infer the locality of the lesion, which is the anatomy, and then by a distinct and separate investigation endeavour to identify the character of the change in the nutrition, which is the pathology.

Convulsions.

We may now try to follow Hughlings Jackson in the study of convulsions with which his name will ever be associated. The fundamental ideas are that all nervous action is of the nature of a discharge and that movements are represented in the cortex of the cerebral hemispheres as well as in the lower centres, and can be excited by discharge taking place in regions of the cortex, destruction of which does not give rise to paralysis. The term discharge is borrowed from electrical science, but its essential character is chemical and of the nature of an explosion, a sudden readjustment of molecules with liberation of energy. There is accumulated in the nerve centres a substance having what I called many years ago chemical tension, in virtue of which it is ready to explode on the application of an appropriate stimulus. In normal nervous action, sensory or motor, the liberation of energy, or discharge, is orderly, limited in amount and strictly adjusted to the stimulus. In convulsion the discharge is sudden and excessive, and disorderly, the seat of the abnormal discharge determining the starting point and course of the convulsive movements, its suddenness and excess, their violence and extent. Suddenness is an essential feature of the convulsive discharge, and it is largely in consequence of its suddenness that the ab-

normal discharge is not limited to the unstable cells in which it begins. The violence of the explosion in them excites discharge of all the cells within reach, not only downwards toward the muscles, but possibly upwards and certainly collaterally. In general convulsions, in which the initial explosion is in some part of the highest centres in one hemisphere, its effects are propagated to other parts of the same hemisphere, and to the opposite hemisphere, as well as downwards. The study of convulsions, therefore, becomes extremely important, not simply from the point of view of verifying their conformity to a given type, but as departures from the normal functional activities of certain nerve centres. The starting point, march, and order of the movements must be precisely noted. It was through this study of convulsions that epilepsy was rescued from the category of so-called functional nervous diseases—functional, that is, in the sense of having no recognised pathology or reasonable explanation. From the fact that an undue degree of vascularity was found in the bulb in epileptics it was inferred that this part of the nervous centres was the seat of the disease, a conclusion supposed to be confirmed and rendered practically certain by the experimental evidence that convulsions were possible when the cerebral hemispheres had been removed. A view so supported and in accord with the generally accepted idea that the cerebrum had no motor functions was not easily overthrown, and it is Hughlings Jackson's incontestible merit to have subverted it, and substituted the doctrine now held with regard to epilepsy. The wedge driven into the old theory was the demonstration that unilateral convulsions of the kind now appropriately known as Jacksonian epilepsy were due to a lesion in the cortex in the Rolandic area, and this it should be understood took place before Fritz and Hitzig's experiments had shown that the electrical irritation of these gyri gave rise to movements. As has been already stated, it was a case of right unilateral convulsion followed by temporary defect of speech which suggested his special investigation of epilepsy. The convulsion must, he inferred, have been due to some affection of the same region, destruction of

which produced permanent loss of speech. In both the cause was embolism, in one case giving rise to a destructive lesion, to instability in the other.

Epilepsy.

One of the great lessons we have learnt is that in approaching the scientific study of epilepsy—and the same may be said with regard to all nervous disease—we must be intent not on noting the conformity of the attacks to a type, but on a precise observation of the departures from normal modes of action which they exhibit.

Another is that epilepsy presents exactly the same problems for investigation as other diseases of the nervous system and the same basis of classification, physiological, anatomical, and pathological—the deviations from normal function presented by the symptoms, the seat of the lesion —the particular nervous centre, affection of which gives rise to the abnormal manifestations—and the nutritive or structural change which has taken place in the nerve centre and which underlies the liability to the special form of functional error.

The idea of epilepsy which we owe to Hughlings Jackson is that epilepsy is the occasional, sudden, excessive, violent discharge of some nerve centre, sensory or motor.

The immediate cause of the paroxysmal tendency is instability of the explosive constituent of the nerve centre in which the initial discharge takes place and it is conceivable that there may be, on the one hand, excessive formation, or, on the other, defective quality of the explosive material. In the latter case the constitution of the molecule may remain the same, while its composition is altered by the substitution of an element or molecule of lower affinities. Studying the conditions under which instability arises they are best illustrated by the lesions in the Rolandic convolutions which give rise to Jacksonian epilepsy. With or without destruction of nerve elements there is congestion, an excess of blood in the capillaries with retarded motion and consequent modification of the nutrition of surviving cells. The chorea

of acute rheumatism associated with capillary embolism furnishes another illustration of instability of nerve-cells induced by interference with the circulation in the nerve centres. When, as in ordinary epilepsy, no morbid change is apparent, it must be assumed that the nutrition of the gray substance is modified so as either to give rise to excessive production of normal explosive matter or to formation of a deteriorated and more unstable variety. According to the degree and character of the exaggerated or perverted nutrition will be the intrinsic epileptic tendency, and its dependence upon or independence of etiological influences or exciting causes with regard to the frequency and violence of the fits. And since there are many nerve centres there are many epilepsies.

They may be divided into (1) epilepsy proper, in which the initial explosion takes place in some part of the cerebral cortex other than Rolandic convolutions; (2) Jacksonian epilepsy; (3) ponto-bulbar epilepsy.

I have here deviated from Hughlings Jackson's nomenclature. He could not perhaps employ the term Jacksonian to designate the unilateral convulsions beginning without loss of consciousness, which, in his hands, have thrown so much light on epilepsy and on the physiology of the cerebrum; at any rate he has never done so, but has always called them epileptiform. We need the term epileptiform, however, in general medicine as a ready method of indicating the general convulsions, which may occur in uræmia and from numerous other causes. It is not, again, strictly accurate as applied to convulsions which differ essentially from those seen in epilepsy. The appellation Jacksonian is generally accepted, and it has the advantage of a definite connotation, which obviates the necessity of falling back on the word "fits," while it is a permanent expression of the homage which the neurological world delights to render to Hughlings Jackson. It was a happy inspiration which led Charcot to employ this designation.

Pursuing the ideas indicated, it follows that the hemispherical cortex outside the so-called motor area being the seat of the complex activities which are concomitant with

the highest mental operations, sudden loss of consciousness without warning would indicate discharge of some part of these convolutions. If motor phenomena predominated at the outset, *e.g.*, a pirouette of three turns, as in a case under my care, the starting point or initial discharge would be in the anterior part of the hemisphere; if sensory coloured vision, stunning noise, a foul odour, the initial discharge would be posterior. The occurrence of convulsions in *grand mal* is determined by the suddenness and violence of the discharge or explosion, which is such as to overcome the resistance of centres in the same and in the opposite hemisphere, discharging them.

When there is an aura this will be the first outside indication of the commotion taking place inside, and will afford a clue to the particular centre which is the seat of the disturbance.

A feature of fully developed epileptic paroxysms is the unconsciousness or coma which follows the convulsion. This represents paralysis of the highest centres presumably from exhaustion. Whatever the cause, however, exhaustion or other condition, coma is paralysis; from another point of view it is a temporary insanity—amentia.

In *petit mal* it is still in some part of the highest cortical centres that the discharge takes place. From the feebleness of the explosion, however, or perhaps in some cases from its slowness, it is only collateral cortical centres, in the same or in both hemispheres, which are discharged, and the explosion does not take effect on the lower centres except perhaps, on a few centres for delicate movements such as rolling of the eyes, twitching of the face or fingers. These, which are genuinely convulsive in character, must not be confused with such actions as smacking of the lips and swallowing, which are motor reflexes from discharge of the gustatory or olfactory centre.

An attack of *petit mal* may be followed by maniacal excitement or by highly complex purposive actions, often very absurd, or by temporary mental confusion. These are not a part of the fit, but are consequences due indirectly to paralysis of the centre in which the fit started, to partial

loss of consciousness, with suspension of the influence of some one or more of the highest centres, and unbalanced or uncontrolled or excessive functional activity of collateral or lower centres.

An interesting set of epilepsies, both *grand* and *petit mal*, has been called the uncinate group of fits. These cases are characterised by a warning relating to the digestive system, a foul odour, a crude sensation of taste, an epigastric sensation, or by reflex movements excited by discharge of the gustatory centre, such as smacking the lips, tasting movements of the tongue, champing, spitting, or swallowing. The localisation is not merely suggested by the fact that the uncinate lobule is the olfactory and gustatory centre and by paralytic counterpart to the spasm, but has been verified by *post-mortem* evidence, in one case a focus of softening having been found in the uncinate gyrus, in others tumours involving the anterior part of the temporo-sphenoidal lobe. The comparatively slight movements have a very important significance as they determine the epileptic character of light attacks which otherwise might seem to be trifling.

A curious fact is the frequent association of intellectual or emotional affections with uncinate fits, the dreamy state, mental diplopia, a sense of fear or horror. This dreamy state, which assumes various forms, is not an aura in the same sense as a crude sensation, but is the effect of a slight cortical discharge. The mental diplopia or double consciousness may possibly represent a discharge which affects one hemisphere only, not reaching the other, and there may thus be normal consciousness in one half of the brain and a slight defect of consciousness in the other.

Vertigo of a sudden fulminating character must be considered to come within the comprehensive definition of epilepsy, even when primarily ocular or auditory, as well as when more strictly epileptic.

Migraine may perhaps be regarded as a sensory discharge of cell-elements in the cuneal region of the cortex.

(2) In Jacksonian epilepsy the lesion, which is usually of a coarse character, thrombosis, syphilitic disease, tumour, is

definitely localised in the Rolandic convolutions. The distinctive feature of this form of epilepsy is that the convulsion begins unilaterally and precedes loss of consciousness, should this occur. While the lesion is obvious and often extensive, the fits are not to be attributed to its destructive effects, but to the modified nutrition and exaggerated instability of neighbouring nerve-cells resulting from the interference with the capillary circulation.

With regard to Jacksonian epilepsy, it is only necessary to call to mind that the convulsion starts in the part of the limb or face which is most employed in fine or complex movements, the hand, and in this the thumb and the index-finger, the parts about the mouth in the face, the great toe in the foot; that it may be (1) limited to these parts, or (2) extend to the whole of the limb without loss of consciousness, or (3) spread to other limbs, in which case the march of the convulsion is upwards in the limb first affected and downwards in the others. Loss of consciousness usually attends such extension, and when the original discharge is sudden and violent the sequence of events may be so rapid that the fit simulates a true epileptic attack.

More or less temporary paresis of the limbs in which the convulsion began follows the attack and sometimes a certain degree of anæsthesia. It is noteworthy that there are never mental phenomena.

An interesting fact of far-reaching import is that convulsion beginning in the hand or foot can often be prevented from travelling up the limb by a ligature or by powerful friction. A powerful sensory impression inhibits the extension of the cortical discharge. The subsequent paresis is apt to be intensified when this has been done.

While epilepsy proper and Jacksonian epilepsy are due to explosive discharge of cortical and cortico-central centres, it was inferred that there might be epilepsies from discharge of bulbo-spinal centres and that these would exhibit special features. Experiments on animals had shown that convulsions might occur when the cerebral hemispheres had been removed, even in the case of the remarkable Brown-Sequard convulsions provoked by irritation of the fifth nerve after hemisection of the cord in the guinea pig.

At first bulbo-spinal epilepsy was considered to be represented by laryngismus, but other forms have been identified.

Several cases have been recorded resembling those artificially produced in guinea pigs. In one brought before the Medical Society and published in the *Lancet* for February 2, 1895, a boy of 7 fell down in a fit whenever his head or face was unexpectedly touched, but not if he knew beforehand. He turned red, looked vacant, his respiration stopped, his eyes were turned to one side, and he would fall if not caught. In some attacks he screamed. It must be specially noted as belonging to the ponto-bulbar seat of the disease that arrest of respiration was a marked early feature of the fits, and it was observed also that he was drawn down by muscular action, *i.e.*, by contraction of the trunk muscles, rather than fell. He remembered nothing of the attacks.

Another form of ponto-bulbar fit is described in BRAIN, Spring, 1902, by Hughlings Jackson and Dr. Douglas Singer. The attacks were frequent and began with simultaneous tonic spasm of the muscles of the neck on both sides, back and front, of the jaws, of the face on both sides, while the thorax was fixed in a position of partial expiration. After a few seconds there was clonic spasm of the same muscles, there were no movements of arms or legs, no change of colour, no sweating. At another stage fits beginning with fixation of the chest involved the muscles of the face, chest, body and upper limbs; in the arms the order in which the muscles were involved was from the shoulder downwards.

Cerebellar epilepsy may perhaps be represented by the sudden opisthotonic spasms which have been observed in some cases of tumours of the cerebellum.

EPILEPSY—PATHOLOGY.

There still remain to be given Hughlings Jackson's views on the pathology of epilepsy. They are that, just as certainly as Jacksonian epilepsy is due to a lesion in the Rolandic gyri, so are epileptic attacks proper the result of a deviation from normal nutrition in some part of the hemi-

spherical cortex other than these convolutions. That the altered nutrition is local, and not vaguely distributed over the entire hemisphere, is shown by the fact that in a given case of epilepsy the fits practically always resemble each other, more particularly in the most important point, their mode of onset. The effect of the nutritional error or morbid change is, as has been already said, instability of a greater or lesser number of nerve-cells, either from excessive formation of the normal explosive constituent, or, as I think more probable, from the formation of a lower grade of explosive matter, which is more unstable. It is possible that the morbid change is primarily and inherently in the nerve elements themselves, but more probably it is a secondary effect of some affection of the non-nervous structures which enters into the construction of the nerve centres. Circulatory conditions certainly play an important part, and the distribution of the altered nutrition seems to follow vascular areas. There are undoubted illustrations of instability from interference with the blood supply, by capillary embolisms in certain forms of chorea and in the congestion around coarse lesions in the Rolandic convolutions. Blood moves more slowly and is detained longer in the affected area. It may be assumed also that in epilepsy caused by thickening of some part of the cranial vault or by pachymeningitis, the cortical circulation is retarded by the pressure. No change may be discoverable by the microscope.

The exciting cause of individual paroxysms is often cardio-vascular, as is suggested by the frequency of epileptic fits during the night and in the early morning, when the circulation has run down. In other cases the intestine or uterus, or other organ or sentient surface, may act the part of an epileptogenous zone.

Evolution and Dissolution.

While the establishment of the motor endowment of the Rolandic area of the cortex by means of experiments performed by disease, and his elucidation of the true nature of

epilepsy are perhaps Hughlings Jackson's greatest and most permanent achievements, his theory of the nervous system as an example of a great evolutionary process and of nervous diseases as examples of dissolution is the most original and comprehensive.

Another keynote may here be quoted:

"Every nervous disease being a flaw in one great evolutionary system demands consideration as wide as we can make it with precision."

From this point of view no symptom is devoid of significance.

The relation between the nervous system and the body generally is characterised as representation. Every part is represented in the nerve centres.

Three levels of evolution in the nervous system are described, exemplifying von Baer's law of progress from the general to the special in function, and from the simple to the complex in structure. The lowest level is constituted by the cord and bulb, by those centres in which the representation of impressions and movements is direct and comparatively simple. We must always bear in mind the acute and profound distinction that it is movements, not muscles, which are represented, impressions, not sentient surfaces. The motor nerve centres do not concern themselves with the instruments; they represent chords, not notes.

While the representation of impressions and movements, or, as we may now say, sensori-motor processes in the lowest level, is direct, the range of the processes may be very extensive. The cardiac and respiratory rhythms are organised in this lowest level at birth, and with them such highly complex respiratory acts as crying, sneezing, coughing. So, again, are the instinctive act of sucking and the reflexes by which food is carried along the intestinal canal, and those by which the bladder and rectum are evacuated. Other illustrations of highly complex and extensive sensori-motor actions organised in the lowest level at birth are the closing of the fingers when the palm of an infant is tickled, and the extraordinary prehensile power which is exhibited in the infant's hands when a finger is placed in each, the grasp being such that

the child can be raised from the ground. There must here be in action not only the flexors of the finger and thumb, but the synergic extensors of the carpus which Dr. Beevor described in his Croonian Lectures.

However complex the movements originating in the lowest level may be, they are, so to speak, automatic, the response to an impression is definite and constant, and, as an impression increases in force and the motor response becomes more extensive, the radiations by which the afferent energy is distributed follow definite lines. The sensori-motor apparatus is organised, the sensori-motor action is automatic.

Since this lowest level is employed by higher centres, there run longitudinally in the cord and bulb not only intrinsic fibres connecting the different lowest level segments, but fibres, afferent and efferent, which connect all parts of the cord and bulb with the cerebrum and cerebellum, and, inasmuch as they do not form part of the mechanism of the lowest centre they may be called extrinsic.

The middle evolutionary level is constituted in Hughlings Jackson's scheme by the central ganglia and the convolutions in immediate relations with them. On the motor side these have been believed to be the Rolandic convolutions posterior as well as anterior to the sulcus, but this opinion may have to be revised in view of the recent experiments of Sherrington and Grünbaum, which seem to restrict motor function to the gyrus which ascends in front of the sulcus.

In this level, impressions from every sentient surface or organ and movements effected by all the muscles of the body, voluntary or involuntary, striped or unstriped, skeletal or visceral, are again represented, or rather, as Hughlings Jackson expresses it are re-represented, and not movements only, but glandular activities and thermal regulation must be thus represented.

The impressions which reach this level are re-representations of impressions made directly upon the lowest level, and the movements are re-represented by combinations of the motor representations existing in the lower level.

Movements best lend themselves to illustration of the

higher stages of evolution, and there are:—(1) increasing complexity—representation of a greater number of different movements; (2) increasing specialisation—representation of movements for more particular duties; (3) increasing integration—representation of movements of wider ranges of the body in each part of the centres; (4) more numerous intercommunications of the units (co-operation).

The automatism is less distinct, the mechanism less definitely organised. And, as is said by Herbert Spencer, "The cessation of automatic action and the dawn of volition are one and the same thing."

Elsewhere (Croonian Lectures) he speaks of evolution as (1) A passage from the most to the least organised—from centres comparatively well organised at birth to those, the highest centres, which are continually organising through life. (2) A passage from the most simple to the most complex. (3) From the most automatic to the most voluntary. If we take most organised to apply to the structural arrangements, most reflex would describe the functional action.

As an illustration of the increased complexity and less definitely organised automatism of movements represented in a higher level, we may take the numerous and complex movements of the hand, fore-arm, arm, shoulder, and trunk, which enter into such an action as the raising of a glass to the lips. Each of them is represented individually in the cord, and they are collectively re-represented in a complex and coördinated way in the central ganglia and Rolandic convolutions. Then as this simple act can be accomplished by a great variety of sequences and combinations of movements, so a potentiality of different coördinations and combinations exists in the middle evolutionary level, by means of which the spinal motor centres of the fingers and other parts of the limb can be called into varied but orderly action.

It is in the middle level that visual impressions are, on the one hand, coördinated with tactual impressions so that they come to correspond as to form, size, and distance of objects, and, on the other, are brought into relation with the

motor apparatus of the lowest level, so that movements may take place under the guidance of vision.

Another question presents itself for consideration. It is in the middle level that activities of the nerve centres first become associated with concomitant states of consciousness. Vision implies consciousness. Visual impressions in addition to the complicated associations which they form with the motor side of the nervous apparatus, by means of which they guide movements, are also referred to a receptive centre, their impact on which is associated with the emergence of that element of a state of consciousness which we call a visual perception.

Different doctrines are held with regard to the relation between psychical operations and nervous activities:—(1) That the mind acts through the nervous system; (2) that activities of the highest centres and mental states are the same thing or different sides of one thing; (3) that states of mind or consciousness are different from nervous states of the highest centres, and that while they occur together there is no interference of one with the other. It is this last which Hughlings Jackson accepts. The functions of the cerebral hemispheres are assumed to be the coördination of impressions and movements. It is true that the cerebral hemispheres are the physical basis of consciousness and mind, but this is in virtue of the fact that in them are represented innumerable impressions and movements of every part of the body. It is true again, as Herbert Spencer says, that mind and nervous action are phases of the same thing. We can think of matter only in terms of mind. We can think of mind only in terms of matter. But for Hughlings Jackson states of consciousness or mind are utterly different from nervous states and, while the two things occur together or in parallelism, there is no interference of one with the other but only concomitance. A visual image arises *during* not *from* activities of the eye and brain. There are not four separate faculties, will, memory, reason, emotion, but four artificially distinguished aspects of states of consciousness.

We may not be able to follow him in this high degree of

abstraction but we cannot too constantly bear in mind that in disease it is structure and function of nervous centres with which we have to deal and not mental faculties, that for example, it is no explanation of speechlessness to say that the patient has lost his memory for words.

The highest level which constitutes the physical basis of mind is constituted by the cerebral cortex generally other than the convolutions which have been experimentally identified as forming the middle level (the motor centres in the Rolandic region, the sensory centres in the angular or calcarine gyri for vision, the first temporo-sphenoidal gyrus for hearing, the uncinate lobule for smell and taste). In these convolutions which receive or emit no radiating or callosal fibres, evolution is carried still further, movements and impressions are in them re-re-represented. (1) The complexity, (2) the specialisation, (3) the integration, (4) the intercommunications, are all carried to a higher power. It is here that the final adjustment of the organism to its environment is effected. The concomitant states of consciousness associated with activities of these convolutions will be abstract ideas, flights of imagination, exercise of the higher reasoning faculties, volitions. These expressions are no doubt wanting in exactness and philosophical precision, but they will not mislead, if we bear in mind the mutual independence of states of consciousness and nervous activities.

Hughlings Jackson holds that not only does the highest level of evolution as a whole represent all impressions and all movements, but that each individual part of the highest centres potentially represents all impressions and movements generally, while representing some impressions or movements more particularly. The pre-frontal convolutions he considers to be the highest motor centres, the posterior extremity of the hemispheres the highest sensory centres. Compensation, the gradual recovery of power after paralysis, he explains from a like point of view; movements represented in destroyed parts are represented, though in somewhat different order, in surrounding parts.

Returning now to the consideration of the relations between the different levels, (1) they are subordinate one to

the other from below upwards, the middle level calling into action in various combinations the motor centres of the lowest level and receiving and coördinating different impressions from this level; the highest level in turn receiving the impressions elaborated in the middle level and initiating sensori-motor impulses which are transmitted downwards, and so to speak translated into appropriate movements by the middle and lowest levels.

(2) Each level is a reservoir of energy capable of independent discharge, and the same may be said of every centre in all three levels. The higher levels, however, not only make use of the lower levels by stimulation and discharge of appropriate portions, but they also exercise control over them and inhibit irregular purposeless discharges. This control or inhibition is of the greatest importance when the effects of dissolution in higher levels is considered.

(3) Further the centres in the different levels are not only reservoirs of energy but positions of resistance. They enjoy a certain degree of independence and each level can resist impulses, afferent or efferent, reaching them from other centres, lower or higher. When, for example, we have ideas and imagination and recollections passing through the mind, these faint states of consciousness are the concomitants of slight activities of the highest centres which do not overcome the resistance of the middle and lowest levels so as to be propagated downwards, and do not, therefore, result in sensori-motor actions. On the other hand the highest level is protected from all but the more vivid afferent impressions by resistance to their transmission from the lowest to the middle and from the middle to the highest. New adjustments and combinations of impressions and impulses can thus be made and tossed about from one convolution to another without interference from below. These may constitute an advance in evolution which may even go on during sleep.

This idea of the mutual resistance between the different levels and centres may be extended. All parts of the nervous system, in my opinion, hold each other in mutual tension, and the passage of an impulse, afferent or efferent,

is better represented as a disturbance of an equilibrium of forces than as a transmission of energy. There is a state of mutual tension between the nerve-endings in sentient surfaces and in muscles and the sensory and motor nerve nuclei in the spinal cord as well as between one nerve centre and another.

It is when the effects of dissolution are considered that the insight evinced in the doctrine of evolution becomes manifest and we see the force of the corollary that "every disease being a flaw in a great evolutionary system demands consideration as wide as we can make it with precision, from paralysis of an external rectus up to insanities." The investigation must not only be minute, but precise. From this point of view no symptom is devoid of significance, the order and march of the symptoms as well as their character are important.

The applications of the doctrine of dissolution to the effects of disease of the nervous system are profound and far reaching. Dissolution implies the reversal of the process of evolution, or descent from the least definitely organised, most complex, and most voluntary, towards the most definitely organised, most simple and most automatic.

Dissolution may take place in any of the evolutionary levels. When this is the case the functions of the centre in which the lesion has occurred are suspended and corresponding symptoms which may be called negative are observed. These, however, are not the only symptoms; other symptoms, usually more obtrusive and often infinitely more important, are produced by the activities of other centres, either *A*, unbalanced in consequence of the absence of normally opposing activities, or *B*, liberated from the control of higher level centres, or *C*, intensified by attempts to compensate for the missing function. Illustrations of indirect effects of this kind are among the most illuminating of Hughlings Jackson's contributions to neurological science. A simple but striking one is furnished by paralysis of the external rectus. There is, of course, double vision but this is not all. In the attempt to carry the affected eye outwards a more powerful stimulus comes to the nerve

nucleus from the higher level but this only takes effect on the associated internal rectus of the other eye. This may or may not exaggerate the double vision according as the object remains within the range of both eyes or is visible only to one, but the position of the eyes being judged of according to the strength of the stimulus issuing from the higher level, an erroneous projection ensues, the object is misplaced in consciousness; and the movements of the body and limbs being adjusted to this false position of surrounding objects, vertigo and staggering are produced.

Examples of simple reduction from voluntary action towards automatism are afforded by the ordinary form of hemiplegia in which the arm is most and longest paralysed and of this limb the fingers and hand, the leg less, the face partially, the bilateral more automatic movements being spared. And also by aphasia in which there is loss of intellectual—the more voluntary—language with persistence of emotional—the more automatic language; impairment of pantomime which is intellectual, while gesticulation which is emotional is unaffected.

"Yes" and "no," which can frequently be said distinctly and appropriately are most general and most automatic. While conveying assent to or dissent from a proposition they indicate an attitude of mind rather than an intellectual act.

Other examples are given but they would require explanation.

Coma and unconsciousness indicate dissolution in the highest level extending in extreme instances to the middle level and to the lowest level, with the exception of the cardiac and respiratory reflex centres.

The most interesting and important illustrations of dissolution are those in which a centre in the highest level is paralysed and symptoms arise from the action of other centres in the same level or in another level. Examples are post-epileptic dreamy states, cerebral diplopia, unconscious actions which may be elaborate and quite purposive, or maniacal excitement. Here the centre in which the discharge has started is left exhausted or, for the time being,

paralysed. The active symptoms result from the activities of other centres on the same level in the same or in the other hemisphere, more or less normal, but unbalanced by the centres paralysed, or from uncontrolled activities of lower centres which are habitually under inhibition by the centres whose functions are in abeyance, or from what Hughlings Jackson calls superpositive activities of various centres, which may result from attempts at compensation comparable to the over action of the internal rectus of one eye when the external rectus of the other eye is paralysed.

Insanity becomes much more comprehensible when considered from this point of view.

But we have to speak of insanities just as we have had to speak of epilepsies, and among the insanities must be included not only permanent or durable mental aberration, but such conditions as drunkenness in its various stages, the delirium of fever, coma, or unconsciousness, however produced. We are thus able to make use of post-epileptic nerve disturbances in the elucidation of insanities.

Speaking of them comprehensively there is dissolution, general or local, in the highest levels of evolution, and the depth or degree of dissolution will vary as well as the situation and extent. This is attended with a corresponding degree of paralysis, which, supposing the dissolution to have taken place in the highest level, will mean loss or impairment of the reasoning power, of the finest emotions, of the adaptation to present surroundings. But this paralysis of centres in the highest level will leave unimpaired and uncontrolled collateral and lower level centres, and their activities, normal or intensified, will be responsible for the active delusions and insane actions which constitute the insane manifestations.

Dissolution going deeper will be attended with greater loss of mental power and fewer evidences of functional activity of lower levels till we arrive at dementia.

Drunkenness in different degree illustrates the whole process of general dissolution gradually reaching deeper layers. Alcohol, like the anæsthetic gases, affects the nervous centres from above downwards. At first the

paresis of the highest level impairs the judgment and self control, the man is regardless of his surroundings, this is the negative side of his condition. On the other hand he is garrulous, in high spirits, excitable, easily moved to mirth or anger. In this we have the play of lower centres no longer under due control, or he may be boisterous and quarrelsome or amatory. The effects of alcohol on different men are very different. This may, in part, be due to greater susceptibility of one centre in the highest level to another in different individuals, but more commonly it is the underlying essential character which is laid bare and in *vino veritas*. As the dose of alcohol is increased the dissolution goes deeper, the man becomes stupid and maudlin and fails altogether to appreciate what is going on around him, his speech is confused, his utterance thick—all indications of a further degree of paralysis. He is no longer garrulous and argumentative but probably repeats the same thing over and over again; his emotions may break loose and he may become the subject of uncontrollable laughter or weeping. The lower centres still capable of functional activity are, even when set free from control, only competent to the production of general and automatic actions. Carried still further the alcoholic poisoning reaches the stage of coma or stupor, the man is dead drunk. The centres of all the levels are paralysed except those in which the cardiac and respiratory reflexes are carried on—there is almost complete dissolution.

There would not be time, even were our knowledge ripe for it, to discuss the insanities due to local dissolutions. One suggestion only need be mentioned; that general paralysis of the insane is due to dissolution affecting the prefrontal convolutions, melancholia to dissolution affecting the posterior part of the brain.

In all dissolutions the rate at which the functional efficiency is abolished will greatly influence the symptoms which result. A cortical change, which, taking place suddenly, gives rise to acute mania, would have no such effect if brought about slowly.

The form and degree of insanity arising from a given

depth and seat of dissolution, again, will vary with the original character of the system of the nervous system in which it takes place, and the degree of evolution which it has attained through education and circumstance.

The factors of the insanities then are:—

(1) Dissolution, varying in situation, extent, and depth, of the highest nerve centres.

(2) The person who has undergone this dissolution of his nerve centres, whether a child, adult, or aged individual, whether originally clever or stupid, educated or non-educated.

(3) The rate at which the control of the highest centres has been withdrawn.

(4) The influence of local bodily states and of external circumstances.

The position of the senses of hearing and vision in the evolutionary system has not been fully discussed by Hughlings Jackson, and the question is a difficult one. The nucleus of the auditory nerve in close relation with the nuclei of the vagus, trifacial, &c., in the floor of the fourth ventricle, obviously belongs to the lowest level. Its lowest level sensori-motor relations seem to be, however, extremely simple. A loud noise provokes a general start, but this is common to any violent impression; there may be an automatic turning of the head and ears in the direction of a sound, and there is the adjustment of the tension of the membrana tympani to sounds of different pitch, which are practically all. A very remarkable fact, the significance and object of which must be important, but are not yet fully comprehended, is the close association between the cochlear or auditory nerve proper and the nerve from the semi-circular canals, which is concerned in the equilibration of the body. Hughlings Jackson points out the impulse to dancing and other rhythmical movements excited by music, which may be almost irresistible, as in some way an effect of this association, but there must be something further.

It is doubtful how far vision can be said to belong at all to the lowest level. The only automatic responses to visual impressions are the pupil reaction to light, the accommodation to near and distant vision and the quasi-involuntary following of objects by the eyes.

The geniculate bodies and corpora quadrigemina which form the apparent terminations of the optic tracts cannot be said to belong to the pons, even if they could be considered to be the nuclei of the optic nerves, which is more than doubtful, and it must be remembered that the optic ganglia in the articulata are supra-œsophageal.

The retina is a combination of peripheral and central structures; while its inner layer in contact with the vitreous is a highly specialised portion of the integument, its outer layer is formed from a vesicular protrusion from the mid brain.

Vision, as a part of the higher evolutionary level, has two distinct relations. The greater part of the most purposive movements are performed under the guidance of visual impressions and visual images are the starting point of intellectual operations. These, in my judgment, must be held to be quite distinct functions. A visual image implies a state of consciousness, the guidance of movements may be effected without emergence in consciousness.

While vision directs movements, movements of every possible kind can be organised independently of sight, as is seen in the case of the blind. We cannot, therefore, say that visual cell groups arrange and control the motor cell groups in the higher level, by means of which the elaborate combinations of movements are executed. These higher level motor cell groups must be organised by higher level sensory cell groups re-representing impressions from the sentient surfaces, muscles, joints, &c. Visual sensory impressions thus find them ready for employment. But granting this, fibres must still pass from the visual centre to the motor cell groups to call them into action which will be efferent fibres of the optic sensori-motor apparatus.

A very important correlation must be effected between tactile and visual impressions in order that the representations and re-representations received from the two sources may correspond. Here again on the sensory side re-representations of tactual impressions must be antecedent to visual representations. Accurate ideas of form and size are formed by the blind, and visual impressions must be

interpreted by the antecedent tactual impressions. Where exactly and by what cell and fibre mechanism this interpretation is effected has not been made clear. Do the tactual centres report direct, so to speak, to the visual centres, or do both report to a higher centre, the primary visual impressions being of a crude character? Or is the correlation effected by fibres passing from one centre to the other?

In the formation of tactual representations movements play an extremely important part, and Hughlings Jackson considers that movements enter into visual representations, form and size being derived from, or representative of, motor activities, colour only being sensory; I cannot altogether follow him in this interpretation of the phenomena of vision. The dominance of the sensory side of the nervous system is not adequately recognised. I cannot argue the question out here, but I may repeat that in my opinion there is a distinction between vision as a guiding sensation for movements, and visual images as the substrata of intellectual operations, and I may add that I regard tactual impressions as being translated into visual for intellectual employment by a process which is at any rate analogous to evolution, visual images re-representing middle-level tactual impressions.

We can only obtain an insight into the mechanism of the highest level through speech, and here we have to rely entirely on the experiments performed by disease. Experiments on animals, however, while they can furnish no direct evidence with regard to the higher cerebral functions in man, afford information of the greatest value and importance. It becomes increasingly difficult when we are dealing with nervous activities, which have as their concomitants mental operations properly speaking, to avoid the use of psychical terms in a physiological sense, but it must be understood throughout that the activities of the highest level are regarded as sensori-motor processes.

I must now attempt to place before the society my own interpretation of Hughlings Jackson's theories of the construction and action of the nerve centres. I am in entire

agreement with him as to the essential nature of epilepsy, and I accept unreservedly in principle his theory of evolution and dissolution with the light it throws on insanity. But for myself I can only think of nervous action in terms of cells and fibres, and I have to endeavour to reduce the refined conceptions embodied in this theory to a cell and fibre mechanism. I must further be allowed to speak of cells and fibres and not of neurones, not only because I am more familiar with them as the structural nervous elements, but because I cannot accept all the implications bound up with the neuron theory or nomenclature.

The cells and fibres are there, and the gray substance constituted mainly of cells is the seat of nervous energy and of intercommunications, and whether the explosive material is formed in the cells or matrix, and whether the intercommunications are effected by cell processes directly or through synapses I am not concerned to discuss.

In the elucidation of the complex problems presented by the physiology and pathology of the nervous system the line of progress has always been to interpret structure by function and not to infer function from structure. To depart from this course, which is that which has been followed with such signal success by Hughlings Jackson, and to erect a theory based upon structure and to make this theory the starting-point of inferences bearing upon function is to open the door to confusion and fruitless discussion. The term neuron is a convenient expression for a cell with its processes and cylinder axis, but some of the theoretical inferences from this idea are in my opinion untenable. The individuality of the neuron, for example, seems to me to be inconsistent with what is seen in the reunion of a divided nerve, the junction of motor fibres central and distal, and of distal with central afferent fibres, we cannot suppose that each sensory and motor neuron in the proximal part of the nerve picks out its own continuation in the distal part, and yet when the recovery of sensation is complete, localisation of an impression is accurate and there is no confusion of movements. The difficulty is increased if a gap in the nerve has been filled by transplantation. Still more difficult is an explanation of the remark-

able experiment of Prof. Robt. Kennedy, related in *Proceedings Royal Society*, lxvii., p. 431, January, 1901. In the right fore-limb of a dog the central segment of the divided musculo-cutaneous, median, and ulnar nerve was united with the peripheral segment of the musculo-spiral, and *vice versa*. Recovery of function took place in about the same time as after simple division of the nerves, and the dog regained perfect use of the limb in walking and running, gave its paw when asked, and used it in holding a bone; the cortical centres were interchanged, stimulation of the extensor centre gave flexion, and of the flexor centre, extension and flexion.

In adopting Hughlings Jackson's idea of nervous representation of movements and impressions, I have considered it more accurate to speak of representation *in* the nerve centres rather than *by* the nerve centres. The representation of movements then in the spinal cord will be groups of nerve-cells arranged *ad hoc*, not the nerve nuclei of individual muscles. This important distinction as enunciated by Hughlings Jackson must always be borne in mind, but isolated movements are rare, and there exists a mechanism in this lowest centre by which the cell groups representing individual movements are associated for combined action to any required extent. The cell groups representing movements must often be very large, for example, the cell group representing a firm grasp of the hand will comprise those representing the synergic action of the extensors of the carpus. While a given movement will always be represented by the same cell group, it does not follow that these cells cannot enter into other combinations representing other movements. The same cells may be differently grouped for different movements, and the cells of one group may form part of a totally different group.

It may be assumed that sensory impressions will be represented by similar groups of nerve-cells in the posterior gray cornua. A noteworthy difference, however, exists between afferent or sensory impressions and motor efferent impulses; sensory areas overlap in the skin, and the conduction of ascending sensory currents in the spinal cord is much more diffuse than the conduction of descending currents to the anterior gray cornua.

An important preliminary question arises—how are these movement-representing cell groups formed? Many of them exist at birth, those which subserve the cardiac and respiratory reflexes for example, and such respiratory acts as sneezing, coughing, crying; those again which are employed in sucking, swallowing, and in the extraordinary prehensile grasp of the infant. But other groups representing movements are formed by a process of education as the child learns to walk and use its hands for various purposes, or as the workman learns to handle his tools. Now all movements are primarily a response to sensory impressions, and are performed under their guidance. The first nerve filaments which appear in the lowest organisms, have for their object to render the motor response to surface impressions more ready and more exact, and this response would be of no service in adjusting the organism to its environment unless it were directed by the guiding afferent impression. It is obvious that the sensory is the dominant side of the nervous system. This dominance of the sensory over the motor side of the nervous mechanism extends to the highest level of the most fully developed nervous system. It is demonstrated by such evidence as the striking and important experiment of Mott and Sherrington, the division of all the posterior nerve roots of the upper extremity in a monkey. The limb was left flaccid and helpless, the animal made no attempt to use it, and seemed unconscious of its existence.

In tabes, again, the degeneration affects the sensory or afferent side of the cord, the posterior root-zone, and the disorderly movements of the limbs are due, not to any motor paralysis, but to loss of the afferent sensory guiding impressions. These direct guiding sensory impressions can be in a measure replaced by visual guiding impressions.

The interpretation I put upon the interesting experiment of Prof. Robert Kennedy already mentioned, in which interchange took place between the flexor and extensor cortical centres both for volitional use of the limb and in response to stimulation as a result of crossed reunion of the flexor and extensor nerves, is that the sensory side of the nervous

system dominates the motor side, and that impressions on a particular sensory area determine the employment of the corresponding motor centres even in the highest level. The process would begin in the cord. The immediate effect of transposition of the divided flexor and extensor nerves would be, as reunion took place, to bring the flexor muscles into relation with the cell groups in the anterior gray cornua, which previously animated the extensors, and in like manner the afferent fibres from the flexor aspect of the limbs would be put in communication with the sensory cell groups previously receiving fibres from the extensor surface. A reflex circuit from skin to muscle would thus exist, an extensor cell and fibre mechanism being employed for a flexor reflex. This apparatus, now flexor, will be in communication with the extensor sensori-motor centre of the higher level, and a flexor response to electrical stimulation of the cortical centre is only what we should expect. What is extraordinary, however, is the re-education of the extensor cortical centre for volitional flexor use and *vice versa*; this must be through sensory impressions, and it is interesting to note that while the flexor response to stimulation of the extensor cortical centre was simple, the extensor response to stimulation of the flexor cortical centre was confused, the afferent musculo-spiral fibres being simple as compared with the afferent fibres of the musculo-cutaneous median and ulnar.

We are apt to lose sight of the fact that sensory nerve endings or beginnings must liberate energy in the act of sending an impression to the cord; there must be a discharge exactly as in the nerve centres. We see in the retina a most elaborate specialisation of nerve endings for the reception of luminous vibrations, the nerves of the skin must equally be specialised for the translation (transformation) of thermal vibrations and of contacts of various kinds into appropriate sensory impression.

The recognition of the dominance of the sensory side of the nervous system I regard as of primary importance.

The structural representation of a guiding sensory impression in the cord will be a group of sensory cells in

which the posterior nerve roots end, dominating the corresponding motor cell group with its efferent fibres, the sensory and motor cell groups being connected by processes. This is the unit of the lowest level. Simple units will be combined for the execution of movements of larger extent, as is illustrated by the extension of a reflex response as the stimulus increases in intensity. The lowest level consists of a chain of such sensori-motor units from the lower end of the cord to the nerve nuclei in the floor of the fourth ventricle united by intrinsic fibres of different lengths and connected by other fibres, which may be called extrinsic, with the centres of higher levels, and it forms a complex and efficient mechanism. How complete the lowest mechanism can be is shown by the headless frog, which rights itself when turned on its back, pushes away an irritant with its foot and, if the foot first used is cut off, will employ the other. I do not think the human cord is inferior in its essential endowments to that of the frog, and although it loses its independence by its subordination to higher centres it retains in my opinion all its potentialities.

An important item in the mechanism of the lowest level is the bi-lateral association of nerve nuclei in the case of muscles of the two sides of the body which always act together and are called into action on one side only rarely and with difficulty, if at all. In the case of bi-laterally acting muscles a commissural association of their nerve nuclei exists which renders the paralysis in hemiplegia partial and which allows movements to cross over to the sound side in hemichorea and hemispasm. It is this bi-lateral association of the nuclei of the occipito-frontalis, the levators and the orbiculars of the eyelids which prevents any obvious paralysis of these muscles in hemiplegia, while it is conspicuous round the mouth, and not any special distribution of fibres connecting the facial nucleus with the higher centres in which the lesion giving rise to hemiplegia has occurred.

In the lateral movements of the eyes and in the rotation of the head the nerve nuclei of the muscles on the two sides, which co-operate, are not on the same level; the part of the nucleus of the third which supplies the internal rectus on

one side acts with the sixth on the other; the part of the spinal accessory supplying the sterno-mastoid on one side acts with the nerve nucleus of the opposite atlo-axoid. The commissural fibres connecting these nuclei respectively therefore, are oblique, and of a certain length instead of being transverse and so short that the two nuclei are practically fused into one. It is this which explains the temporary conjugate deviation of the eyes and head which is often observed after severe hemiplegia; the external rectus of the paralysed side and the atlo-axoid are paralysed; the eyes and head are acted upon by the now unopposed muscles of the sound side and turned so that the patient is looking towards his lesion. This only lasts for a few days, till the incitation, which has been intercepted by the lesion and which, reaching the sixth nucleus and the nucleus of the atlo-axoid, was normally propagated to the third and the spinal accessory, has started from the uninjured hemisphere and found its way round in the opposite direction from the third to the sixth and from the spinal accessory to the atlo-axoid nucleus. The fact that the deviation of the head and eyes occurs together and that it is fugitive makes it clear that this symptom is not due to extension of the lesion to any particular cortical centre. Here, as in the more direct bi-lateral association of nerve nuclei, the commissural fibres, which obviate paralysis by conducting the motor impulse from the uninjured hemisphere to both sides of the cord, conduct the morbid discharge to both nuclei, so that in hemispasm the conjugate deviation of the head and eyes is away from the hemisphere in which the lesion exists. There may, again, be permanent lateral deviation of the eyes in lesion of the pons implicating the commissural fibres between the sixth and third.

The bi-lateral association of nerve centres will be shown to have important applications in the higher levels. I think a further exemplification of the principle exists in the cord, in fibres which may be assumed to extend from the arm centre of one side to the leg centre of the other, or *vice versa*, which have to do with the automatic swinging of the arms in walking and running; which may, moreover, have

to do with the imperfect paralysis of the leg in hemiplegia, and which certainly, in my opinion, give the explanation of the circus movement of the dog instead of hemiplegia in severe lesion of the motor area.

The cell groups organised for movements in the lowest level are made use of by the centres of higher levels. Indeed, to say that existing cell groups representing them in the lowest level are made use of and are brought into action in greater numbers and more varied and more purposive combinations in a higher level, is simply another way of stating that movements are re-represented in a higher level with increasing complexity, specialisation and integration.

But just as movements are represented in the cord by cell groups so will they be re-represented in the next higher level by cell groups. These must be so complicated and their inter-relations so intricate as to defy the imagination, but this is no reason for denying their existence.

Following out the same idea with regard to impressions, they will be re-represented in the next higher level by groups of sensory cells in relation, not directly with fibres of the posterior nerve roots, but with the sensory cell groups in the cord and bulb, which primarily represent impressions and guide movements, and, just as the sensory cell groups arrange and govern the motor cell groups in the cord, so must it be in higher levels, and it must be through the sensory cell groups that the complex motor cell groups are arranged for their duties.

But interposed between the lowest and next level, or superposed upon the point of junction is the cerebellum, which, from its size and extensive relations, must play an important part in the mechanism by which the higher level makes use of the lower. It is connected by its inferior peduncle with the cord, with the central ganglia and cerebrum by the superior peduncle, but the middle peduncle is much larger than the superior and inferior put together and its fibres decussate in the pons, interlacing with the longitudinal fibres of the cerebro-spinal axis and ending in the cells of the gray matter which occupies the interstices between the transverse and longitudinal fibres and no doubt connect them. Both experiment and disease show that each hemi-

sphere of the cerebellum is functionally associated with the opposite cerebral hemisphere and the same side of the cord.

Now, we know that the cerebellum has nothing to do with the initiation of purposive movements of the limbs or with the sensory cell groups which govern them. There is practically left to it only functions which will come into operation in subordination to the cerebrum. One view is that it specially coördinates the movements of locomotion. In my opinion this is a very imperfect conception of the part played by the cerebellum. It is the seat of the elaborate coördinations which are demanded by every movement of both upper and lower limbs. This may be illustrated by an example. A man strikes a blow; the cerebral purposive part of the action is the clenching of the fist and the shooting out of the arm in a definite direction. But the act, to be effective, demands the co-operation of every muscle of the body, the trunk becomes rigid and is thrown powerfully forward, the legs are firmly planted and spring to the effort. This is what is done by the cerebellum. Such an interpretation of its action falls in with Hughlings Jackson's opinion that the cerebellum is mainly concerned with movements of the body as distinguished from the limbs. The trunk muscles must co-operate in every action of the upper limbs as well as in the balancing of the body in station and locomotion in which the lower limbs are engaged.

Hughlings Jackson has observed cases in which tumour of the middle lobe of the cerebellum has given rise to tetanic rigidity of the spine, and he instances a most instructive contrast between the phenomena of Jacksonian cortical convulsions and this condition. In the one, movements unilateral, beginning in the hand, spreading upwards, clonic; in the other, bilateral, as are the usual movements of the trunk, invading the limbs downwards, tonic in character. The unopposed cerebellar influence to which Hughlings Jackson attributes rigidity and exaggerated knee-jerks is another name for the mutual tension which exists between one centre and another.

Where, then, is the first of the higher levels? In this lecture I have followed Hughlings Jackson in speaking of it

as constituted by the corpus striatum and the Rolandic gyri, but the view that I have held has been a very old one—that it was constituted by the thalamus and corpus striatum so that I make four levels, and, while I acknowledge that the evidence of Wallerian degeneration appears to be altogether against me, I am not yet entirely convinced that these ganglia do not constitute a sensori-motor level.

My conception of the relation between the different levels does not admit of continuous fibres from the motor area of the cortex to the anterior gray cornua or of afferent fibres unbroken by cell-network from cord to cortex, whether from the point of view of a mechanism competent for the complicated relations involved, or from the point of view of evolution. It is incompatible, indeed, with re-representation.

If the degeneration seen in the pyramidal tracts after a cortical lesion in the Rolandic region is absolute proof of continuity of the fibres without interruption by cells or communication with cells by branching in the corpus striatum, this view can scarcely be sustained. But if fibres of the corpus callosum are reached by degeneration in advanced stages of lateral sclerosis of the cord, as described by Mott in a recent paper in BRAIN, it is clear that cells do not constitute a barrier to secondary degenerative processes. I do not propose to argue the question, but these great masses of gray matter, by far the largest aggregation of cells which are to be found in the nerve centres, and which seem to form a distinct and natural link between the cord and brain, are apparently left without function. I cannot believe that experiment has said the last word with regard to them and there comes to mind Hughlings Jackson's repeated and emphatic contention that discharge may give rise to movements, when destruction or removal does not produce paralysis, and that the experiments performed by disease may disclose nervous mechanism and function. At one time when hæmorrhage gave rise to hemiplegia the lesion was described as being in the corpus striatum, but the corpus striatum is now deposed in favour of the internal capsule. The internal capsule, however, belongs in great part to the

corpus striatum and thalamus and is largely made up of fibres arising in the cells of the central ganglia and passing between them and the convolutions. The central ganglia seem to me to be singularly suitable as the seat of the complex cell groups, sensory and motor, just considered, and that they must play some extremely important part in the mechanism of the nerve centres is shown by the enormous accretion which they contribute to the fibres ascending from the cord and bulb to the cerebral hemispheres.

Before going further I must devote a short time to the conclusions at which I arrived in an endeavour to trace the course and destination of the fibres which ascend from the cord to the brain, and to obtain an idea of the structural arrangements of the hemispheres.

To this task I devoted two years, and, while I am the first to acknowledge the limitations of the method I employed, which was to follow the fibres in the spirit-hardened brain, I can speak with confidence of such results as I obtained.

Beginning with the crus cerebri we have in it for the first time a separation between the sensory and motor tracts, the pes or crusta, motor, the tegmentum, sensory. In both there are many more fibres than can ascend from the lowest level, including, of course, among these not only the continuation of the cord, but also the crossed fibres from the nerve nuclei in the floor of the fourth ventricle and fibres ascending from the corpora quadrigemina and optic tracts. The additional fibres are contributed by the cerebellum, directly by the superior peduncles, which merge in the tegmentum; indirectly from the gray substance in the interstices of the decussation of the middle crura, which apparently form part of the crusta. The red nucleus in the tegmentum corresponds with the olive in the medulla, and belongs to the cerebellar mechanism.

Following the composite tracts upwards they expand fanwise, the fibres diverging rapidly, and there is a gentle rotation of the plane so that the borders of the two fans become approximately anterior and posterior,

the internal tegmental surface, which is convex, looking upwards, the external, slightly concave, surface looking downwards. Anteriorly, the internal fan overlaps the external and curls round it towards the anterior perforated space in such a way, that Gratiolet speaks of "la partie perforante" and "la partie perforée." If we may be allowed to speak of the caudate body and the lenticular body as component parts of the corpus striatum, the corpus striatum may be said to sit astride of the anterior border of the double fan, while the thalamus, resting on the inner fan below the caudate nucleus, curves round the posterior border, narrowing down into a sort of tail, which forms the collar of the peduncle along the course of the descending cornu of the lateral ventricle. The double fan-like expansion of the crus just described is what has been called the internal capsule, because in its concavity lies the lenticular nucleus. This name is singularly inappropriate, and under the disguise which it affords, the multiple source of the fibres and their afferent, as well as efferent course, escape attention. A better name would be the central expansion of the peduncle. It is here that cerebral hæmorrhage most frequently occurs, and it is to rupture of its fibres by the extravasation that the paralysis is attributed, but there must also be damage to the gray substance lying between the white striations, *i.e.*, to the corpus striatum, and extensive disruption of cell and fibre connections.

What proportion of the fibres, crustal, or tegmental, or cerebellar, continue upwards to the convolutions, what proportion end in the central ganglia, what part is taken by the masses of cells in the thalamus and corpus striatum in the relations between the cord and the hemisphere, is not definitely known. It is, however, certain that great numbers of fibres, afferent and efferent, arise in the central ganglia and pass with the diverging crural fibres to the corona radiata. Large bundles can be seen in the spirit-hardened hemisphere to pass upwards and forwards from the thalamus; the external capsule is formed by fibres which arise from the gray matter of the lenticular nucleus and pass

upwards to the corona radiata as it emerges from between the central ganglia, intersecting its fibres at an acute angle; it is less easy to trace fibres from the caudate nucleus, but they are evidently very numerous. Before the radiating fibres of the two fan-like expansions, at first quite distinct, emerge from between the central ganglia they have become commingled, and a great interlacement of fibres takes place. They are now re-named and become the corona radiata. In the corona radiata it is no longer possible to distinguish between the fibres having different origins, but, while crusta-corpus striatum fibres predominate anteriorly, and tegmento-thalamus fibres posteriorly, it appeared to me that, for the most part, if not entirely they were distributed together, and I could assert with absolute confidence that they passed only to what may be called marginal convolutions—the upper border of the hemisphere, the frontal extremity and the occipital tip, the convolutions around the sylvian, parieto-occipital and calcarine fissures, the gyrus hippocampi and the two great Rolandic gyri. Large areas of convolutions remain which receive no radiating fibres whatever, and these are the very convolutions which are interposed between the simple marginal convolutions of the lower monkeys to constitute the difference between their brain and the brain of man.

Flechsig, by the method of tracing the order of myelination of fibres in the nerve centres of the fœtus, which he originated and has followed out with such interesting results, has more recently reached the same conclusion that considerable areas of convolutions in the cerebral hemispheres are what he calls centres of association, receiving no projection or radiating fibres, and these correspond in great measure with those which I indicated. I may point out, however, that the radiating projection fibres have various sources and in particular that they are efferent as well as afferent. Efferent functional activity will be a necessary consequence of the functional activity of afferent fibres, and of the cortical gray centres, to which they pass, and the fitness for this will involve myelination. A better name for these sensory projection centres, all of which are marginal,

would, therefore, be sensori-motor. The bodily sensibility projection centre, however, as indicated by Flechsig, encroaches upon a part of the inner flat surface of the hemisphere which I found to belong to the longitudinal commissural system of this aspect, fibres arising in the gray matter of the gyri curving downwards and then forwards and running parallel to the corpus callosum.

A further interesting fact is that the fibres of the corpus callosum are distributed to the same convolutions as the radiating fibres, though in very different proportions, and that they connect throughout, the corresponding parts of the two hemispheres, thus constituting a bilateral association. The most abundant distribution of callosal fibres is along the great longitudinal convolution, which receives comparatively few radiating fibres and apparently only in certain parts, while the number going to the Rolandic convolutions seems to be small.

It is probable that fibres of the corpus callosum bend down to form a commissure between the central ganglia; it has, indeed, been asserted that this was its entire object.

When we examine the structure of the individual convolutions, we find that fibres arising in the gray matter of the sides of the sulcus up to the free surface of the convolution curve very obliquely under the bottom of the sulcus to the gray matter of the adjacent gyrus. Some of the convolutions are entirely exhausted by such fibres. In most, a thin plane of fibres remains along the axis of the convolutions, arising from the gray substance at its summit. This axial plane is thickest in the prefrontal convolutions, which are themselves comparatively small, and here also the arrangement of the fibres is most intricate. It is only the summit of the gyrus which receives radiating fibres or fibres associating it with distant convolutions. The association or convolution commissural fibres mostly take a longitudinal direction. In the posterior part of the hemisphere fibres can be seen to curve forwards to form a large mass in the long axis of the hemisphere, which can be followed as far as the posterior Rolandic gyrus. Here they intersect the radiating fibres, and probably many turn aside with

them to end in the Rolandic convolution, but masses of fibres can be seen to emerge in front of the anterior Rolandic gyrus, to be distributed to the frontal convolutions. Along the inner flat surface of the hemisphere is another system of longitudinal fibres, from the parieto-occipital fissure forwards. A smaller system runs in the depth of the Sylvian fissure.

Since the corpus callosum, while connecting symmetrically convolutions in the two hemispheres which receive radiating fibres, is predominantly distributed to the great longitudinal convolution which receives comparatively few, and apparently very little to the Rolandic area, in which so many radiating fibres begin or end, it is probable that it is largely a commissure of the highest level.

Language.

The functional activities of the highest evolutionary level are not open to experimental investigation, and we have to rely on the experiments performed by disease for our knowledge of their physiology and pathology. The most characteristic manifestation of these activities is speech, and it is by study of the affections of speech that a clue is obtained to the mechanism underlying the intellectual operations properly speaking.

Hughlings Jackson has not given us any complete theory of language, but I am not sure that he has not done more towards the elucidation of the problem of speech than those of us who have attempted its solution systematically. He has, at any rate, laid a solid foundation for enquiry and cleared the path of investigation by his fundamental analysis of language, the division of speech into intellectual expression and emotional expression, including in the first pantomime, in the second gesticulation. The unit of intellectual expression is a proposition, the third left frontal convolution being the way out for intellectual expression. There is no such thing as a faculty of language. Language, like all nervous operations, consists in the coördination of impressions and movements and is a sensori-motor process. Intellectual

expression, when followed to its source, merges in the larger term of special movements acquired by the individual; emotional expression consists of inherited movements and is automatic.

This fundamental distinction between intellectual and emotional expression enables us to understand how a man otherwise speechless can give vent to oaths and exclamations, may be surprised into saying "How do you do?" or "Good-bye," can often say "Yes" and "No," the articulation in all these cases being distinct. The threefold employment of the word "No" is instructive; it may be an emotional expression, or an intellectual negative, or an imitative articulation, and cases are not uncommon in which an aphasic patient will answer "No" appropriately and unhesitatingly who cannot repeat the word when asked to do so; in one instance which I have recorded, the patient, after great effort to repeat "No" after me, would give it up with a negative shake of the head, saying "No, I can't."

The views I entertain on the mechanism of speech and thought have been set forth in papers contributed to the *Medico-Chirurgical Transactions*.[1]

Words as movements, like all other movements, are represented in the nervous centres by motor cell groups, and these motor word cell groups will, like all other motor cell groups, be arranged and guided by sensory cell groups, which will in the case of words be auditory. Now words may be merely imitative, may have no symbolic significance, as in the words spoken by a parrot or taught to a child, irrespective of their meaning, or belonging to an unknown tongue. An articulate sound as heard is represented by a given arrangement or grouping of receptive cells in the auditory sense centre; it is imitated or reproduced vocally by a corresponding definite arrangement and combination or grouping of cells in a motor centre, which is effected under the guidance of the auditory cell groups. The motor group when formed calls into action the appropriate motor nerve nuclei concerned in articulation.

[1] Vol. lv., 1872; lxi., 1878; lxvii., 1884.

An articulate sound may further, by association, be made to indicate an external object without rising to the dignity of an intellectual symbol, as when a dog or an infant knows which member of the family is designated by his or her name. There is here a simple association of a visual and an auditory impression.

It will be convenient to consider here the extension of the principle of the bilateral association of nerve nuclei to the higher centres. It leads by an independent and quite different line of argument to the conclusion in which Hughlings Jackson thought he stood alone, that words are represented in the right as well as in the left hemisphere. The principle, it will be remembered, is that when muscles on the two sides of the body always act together, their nuclei situate in opposite sides of the cord are so closely associated by commissural fibres as to be practically one nucleus. Now in speech all the muscles employed are bilaterally associated. In phonation the bilateral association of the laryngeal muscles is absolute, and while the two sides of the tongue and lips are capable of independent action they always act together in articulation. There must therefore be motor cell groups representing words in each half of the bulb, and they will be symmetrical and bilaterally associated.

Not only have we bilateral association of motor nerve nuclei for articulation in the bulb, but, while there is no bilateral association of sensory nerve nuclei in the cord, when we come to hearing and vision the sensory impressions from the two ears and two eyes are, so to speak, fused; there is complete bilateral association.

Now just as the cell groups representing given movements in the cord are arranged by corresponding cell groups, so the cell groups for words will be arranged under the guidance of auditory impressions. At a higher level, again, the motor centre for imitative articulation will be educated by the auditory perceptive centre. But hearing, being bilateral, the auditory perceptive centres on the two sides will be bilaterally associated, and it must follow that the motor centre for imitative articulation is educated in each hemisphere. A further conclusion which seems to me

to be inevitable is that the right third frontal convolution is educated for utterance as well as the left. Motor cell groups for words will be organised in both. It is, of course, difficult to reconcile with this view the specialisation of the third frontal of the left hemisphere only for speech, but this difficulty exists whatever our theory of speech may be, and the employment of one side only of the brain in intellectual expression can never cease to be a matter of astonishment.

The explanation is Moxon's hypothesis that the left hemisphere is predominantly employed in the most voluntary of all movements of the limbs, those of the right hand, and that this has led to the education of the hemisphere for language, which is the highest and most complex of voluntary movements.

The view that verbal cell groups are formed in the right third frontal gyrus endows with special interest the part of the corpus callosum which unites the two third frontal gyri. In my opinion, it is by the commissural fibres crossing from one to the other that aphasia is escaped when the descending fibres from the left third frontal are torn across by a hæmorrhage in the internal capsule. The gray matter not being damaged, speech, organised in Broca's convolution for transmission downwards, crosses over to the right third frontal, where, as has been said, motor word groups already exist, and in this way reaches the bilaterally associated nerve nuclei in the bulb. A certain delay may be required for the establishment of this roundabout route. If in a case of right hemiplegia without aphasia the anterior part of the corpus callosum were divided, aphasia would follow.

The curious mirror writing sometimes seen in right hemiplegia without aphasia seems to afford strong confirmation of the organisation of word groups in the right hemisphere. The right hand being paralysed the patient cannot write. He at once, however, proceeds to write with the left, currently and without practice, but in the opposite direction across the page and apparently illegibly till the script is held before a mirror, when it is seen to be the patient's normal handwriting. The only explanation of this extraordinary accomplishment which seems possible to me is

that concurrently with the education of the graphic motor centre in the left Rolandic area there has been an unconscious imitative education of a coresponding centre in the right hemisphere, which could only be effected through the commissural corpus callosum fibres connecting the two.

Before the employment of words as symbols can be followed I must enter upon a brief explanation of my view of the evolution of ideas, and of the nervous mechanism concerned in it.

As impressions ascend from one evolutionary level to another and from being represented are re-represented and re-re-represented, we have not only the increasing number, complexity, and indefiniteness of these inter-relations, but what may be called an intellectual elaboration. In the infant, while the upward paths in the hemisphere are still undifferentiated, the concomitant state of consciousness will not rise higher than crude sensations of light, sound, contact. A step higher is perception, which for my present purpose I define as the recognition of the external cause of any sensation. The seat of this elaboration I believe to be those of the marginal and post Rolandic convolutions, to which are distributed afferent sensory radiating fibres. They will constitute the visual, auditory, olfactory, gustative and tactual perceptive centres. Each of them will educate its corresponding motor centre. The senses of smell and taste may be dismissed as having practically no intellectual associations, though odours have curious emotional relations. The tactual perceptive centre will organise all purposive actions, especially the varied and delicate purposive movements of the hands; the visual perceptive centre will take part in this intellectual education of the hands and other parts, inasmuch as the movements, though organised tactually, are, for the most part, employed under the guidance of vision. It is argued (Spencer) that "tactual impressions are those into which all other impressions have to be translated before their meanings can be known." Accepting this as true, it is in visual perceptions thus verified that mentation is, for the most part, carried on. Visual ideas stand on a higher intellectual plane than tactual

impressions; they re-represent them and are, in a sense, symbols.

The auditory perceptive centre will educate the third frontal convolution, will direct the formation in this gyrus of word groups, which no longer represent simply imitative articulate sounds, but sequences of words as employed in language. Articulation is not affected in lesions of this centre. We have loss of speech or mistakes in words.

Even now, however, we have only reached a rudimentary stage of intellectual evolution. We have to consider the formation of ideas and their embodiment in words; the employment of words on the one hand as symbols representing elaborated sensory impressions, and on the other as expressive of intellectual motor activities. For Hughlings Jackson, the idea of an external object is a faint image of a previous sensation, and is concomitant with a faint reproduction of the activities in the same cell and fibres, which, when strongly excited, were the anatomical substratum of the sensation excited by the object; the very same cells and fibres which are brought into activity when a sensation of any kind is experienced or an action performed, are engaged in its reproduction in memory. This may be accepted as far as it goes, but the idea, say of an orange, is compounded of several perceptions, colour, form, size, feel, fragrance, taste. It seems to me that we have here a further intellectual elaboration, and that its seat and substratum will be some part of the superadded convolutions. My hypothetical interpretation of the process is that impressions are transmitted from each perceptive centre to a higher cortical centre, where they are coördinated into a complete idea or mental picture of the object. But all these qualities, if we may use the term, or perceptions are suggested or brought into consciousness by the name, that is, a further association is effected in this centre with the name—an arbitrary auditory symbol, which has no relation whatever to any property of the object, but which thenceforward represents it in consciousness. The act of naming, of symbolising by a word, marks an enormous intellectual step, and is the basis of all mental operations. The cortical area, in which the coördina-

tion of the various perceptions and the associations with a name takes place, I have called the naming centre. Another name for it would be the idea centre.

The assumption that such a centre exists and is the starting-point for further intellectual operations, seems to me to be the simplest and indeed the only explanation which my imagination can grasp of the different forms of sensory aphasia. One of these forms is the so-called word-blindness, a case of which I related in the *Medico-Chirurgical Transactions* for 1872, vol. lv.

In this case, however, as in every case which I have personally seen, the word-blindness was only a part of a much wider defect, and applied to such a case the term "word-blindness" lacks precision and is altogether misleading. It was not only that the patient did not recognise printed or written words, even when he had written them himself, but he could not name any object whatever at sight. If a wrong name were suggested, however, he would dissent, and would eagerly point to the object to which the name belonged.

Here the path from the visual perceptive centre to the naming centre had been destroyed. I will not venture to say that the loss of the power to name things at sight is present in all cases of word-blindness, but it is often overlooked. On several occasions when I have been asked to see a case of word-blindness, this wider defect had not been suspected until the test has been applied at my suggestion.

A more complicated form of sensory aphasia is word-deafness, when the path between the auditory perceptive centre and the naming centre has been divided. Here the confusion is much greater. The patient cannot understand what is said to him, and he may or may not know what he himself says.

In other forms, the naming centre itself appears to be destroyed.

I have recorded cases exemplifying each of these lesions.[1]

Carrying out the idea of sensori-motor processes, the correlative of the naming centre will be a propositionising centre, which I take to have its seat in the prefrontal con-

[1] *Medico-Chirurgical Transactions*, lxi.

volutions. This has subordinate to it the two chief ways out for intellectual expression, speech and writing, the articulatory and the graphic. Speech and writing are commonly lost together, and since writing is a later acquisition than speech and is learnt through speech, it was natural to conclude that, in a sense, writing was only a subordinate form of articulate language, and could never be so far independent of it as to be a vehicle for intellectual expression when speech was lost. Examples are, however, on record, and more than once I have seen writing recovered before speech, when both have been lost. This seems to me to imply the existence of a centre which employs both the graphic centre and the way out for spoken words. It is above both and forms the propositions which are transmitted to them for expression. Moreover, educated and intelligent patients, capable of understanding the question, have told me after their recovery, that while speechless, they not only knew what they wanted, but could mentally rehearse the words and phrases which expressed their wishes.

Between the naming centre and its propositionising centre will be fibres, which I believe to be the fibres which pass forward in the axis of the hemisphere from the occipital to the frontal convolutions.

The only parts of speech with which the naming centre will be concerned are nouns, which are the sensory elements of language, together with pronouns and adjectives. All other parts of speech are motor, and these the propositionising centres will supply. I have reported two cases which appear to me to exemplify division of the fibres, connecting the naming and propositionising centres. The patients had completely lost nouns. One of them I saw from time to time and had long conversations with, and for two years he was under the observation of an educated and intelligent nurse and of relatives, who were instructed to be on the look out for nouns in his speech, and were competent to distinguish them. During these two years, while he could say any other words and could communicate his wishes by words and signs, he never said a noun. In the other

instance, I sat by the bedside of a lady who was talking volubly all the time for twenty or thirty minutes, during which she did not utter a single noun, except adverbially. The name or noun centre could not supply nouns to the motor propositionising centre, which was itself in good working order.

On the other hand, in a case reported by Professor Oliver, of Newcastle-on-Tyne, *British Medical Journal*, July 11, 1903, the patient could not put a sentence together, she "could supply all the nouns and many adjectives, but could not put in the verb." The motor side of the propositionising sensori-motor mechanism was damaged. Cases such as this are very uncommon, but it may be because the prefrontal convolutions are so rarely the seat of disease.

In association with the naming centre, which will be somewhere in the posterior part of the hemisphere, will be the convolutions, which are the seat of the nervous activities corresponding to the forming of abstract conceptions, the comparison, ordering and association of ideas, the play of the imagination, which constitute the exercise of the intellectual faculties. These are essentially sensory operations; some of us think mainly in visual images, some mainly in verbal symbols, and the thinking goes on without seeking outward expression. The correlative highest motor mental faculties will have as their instrument the prefrontal convolutions, which will be reached by commissural fibres from the sensory convolutions lying behind the Rolandic gyri.

Innumerable questions present themselves for discussion when the mechanism of speech and thought are under consideration, but there is only one which is of sufficient importance to be alluded to in this lecture. It is the remarkable fact that many aphasics can utter numerals—can count from 1 to 20 when they cannot recite even a fragment of the alphabet—can tell accurately the number of coins shown to them—in one case the numeral would carry with it the name of the coin "five shillings"—can answer questions relating to numbers—can even write figures if not paralysed. There is nothing emotional about arithmetic

and accurate enumeration is not automatic. We cannot fall back on either emotional expressions or automatism as an explanation. To state a number might almost be called propositive. It is possible that the explanation lies in the fact that number is an abstract conception of a quite special kind, which, though formed by enumeration of external objects, becomes independent of them, ceasing to have any relation with visual or other sensory impressions proper, and that the right hemisphere is concerned in this mental operation.

There are interpretations of the physiology of speech in which I cannot follow Hughlings Jackson. He speaks of the left as the leading hemisphere, which is indisputable, and of the right as more automatic, which is probably true, suggesting that in motor aphasia, when the patient while speechless is not wordless since he understands what is said to him, the words are revived in the posterior part of the right hemisphere, and that such recognition of words is automatic because it is in a sense involuntary. I cannot regard the comprehension of spoken words as automatic; it is an acquisition by the individual, as speech itself is, and that the right hemisphere is not concerned in the recognition of language, spoken or written, is shown by the so-called word-deafness and word-blindness, which can be induced by lesion in the posterior part of the left hemisphere.

I am unable again to regard words when employed in thought, especially words expressing abstract ideas, as revived articulatory movements or to consider that motor aphasia is adequately explained as being the loss of articulatory motor processes. It is no doubt true that mental states attend the energising of motor as well as sensory processes, of outgoing as well as incoming currents, but in my judgment words in mentation are sensory symbols; their significance as such is learnt before utterance is acquired.

While I agree with Hughlings Jackson that all parts of the body are re-re-represented in the highest centres, all afferent impressions from sentient surfaces, muscles and viscera, and all movements, skeletal, visceral, vascular,

secretory, and thermal, which are excited by efferent impulses, I do not go with him in his view that each unit in the highest level represents all movements and all impressions, certain impressions or movements specially and particularly, others generally and slightly. As a cell and fibre mechanism this conception raises difficulties which are insuperable.

Certain facts which he thinks cannot be explained on any other hypothesis than universal representation—why in hemiplegia the hand, with its fine purposive movements, always suffers most, and hemispasm of cortical origin always begins in the hand; how compensation with recovery of some degree of function can be effected after destruction of a cortical motor centre if the movements are not represented in other parts of the cortex, are, it seems to me, easily understood on his own exposition of evolution and dissolution. It is only in the highest level that the most purposive movements are represented and it follows that in destruction or discharge of highest level centres they will be the most affected while more automatic movements of the upper limb are represented in lower levels and will be less affected. So, again, a certain degree of compensation may be effected by readjustment of cell groups in the lower levels.

I agree also with Franck and Pitres that convulsions started by a discharge in a limited area of the cortex may be generalised by the lower centres.

It will be understood, I hope, that in these remarks I speak as a disciple not as a critic.

There is no finality in nervous physiology and pathology any more than in other branches of knowledge but it is my unwavering conviction that, whatever further advance is made, the work of Hughlings Jackson will always form one of the corner stones of neurological science. It belongs to the highest evolutionary level of the neurology of the present day and the ideas found in his numerous writings will be important factors in any fuller comprehension of nervous action and function which may be hereafter attained.

The theories of his, which I have brought once more before the Society, are no fantastic hypotheses, having no point of contact with nature or practical application to our work as medical men. They have been projections of the scientific imagination into the unseen, such as are always necessary to the marking out of the pathway for the advancement of knowledge. From their very nature they have conduced to precision as well as minuteness of observation. They have given point and direction to clinical work and so rendered it more valuable, and have inspired and guided investigation

Were they swept away, which, for my part, I do not believe will ever be the case, it would still be emphatically true that Hughlings Jackson has been a pioneer in nervous physiology and pathology.

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ON THE STUDY OF DISEASES OF THE NERVOUS SYSTEM.

BY J. HUGHLINGS JACKSON, M.D.

A Lecture delivered June, 1864.

(*Reprinted from the London Hospital Reports, vol. i., 1864, at the special request of Sir William Broadbent.*)

GENTLEMEN,—"It is easier," Lord Bacon says, "to evolve truth from error than from confusion." If we look back, most of us would say of our mental culture that the great want was not a want of industry, nor of opportunities, but of method. And in the study of disease it is better to have even a mechanical arrangement than to work without a plan. But I hope that the method I am about to submit to you is in some sense a natural system, rather than an artificial scheme. If you find it helps you keep it; if it trouble you reject it. Each has his peculiar mental temperament, and a plan which to one is a help would be a constraint to another. I think the value of my method is that it will prevent your mistaking a complete view of one aspect of a case for a knowledge of the patient's disease. Thus, you may study the physiology of a case minutely and accurately, but this is not the disease. You may study the clinical history and neglect the physiological; this also is an imperfect foundation for thinking of a patient's case. You will see better what I mean after a few illustrations. I will now simply repeat what I have said, that neither the physiological history alone, nor the clinical history alone, is to be taken as a basis for our diagnosis, or as a basis of treatment. It is from a consideration of both, that

we arrive at the natural history and act for our patient's advantage in diagnosis, prognosis, and treatment.

But I must insist on one thing as an essential preliminary in the study of disease, and markedly essential in the study of diseases of the nervous system, viz.: a knowledge of anatomy. Try to explain to anyone who does not know anatomy, from disease of how many different cranial nerves sight may be affected. It would be as hopeful to try to explain to a blind man what the colour blue was like. For we must, in studying diseases of the nervous system, be precise and not merely minute. I believe, and I hope I shall give you some reasons for my belief, that diseases of the nervous system are not more obscure than those of the chest or abdomen. It is the anatomy that is difficult. It is a truism to say that we cannot tell the value of "strabismus" as a symptom in brain disease if we do not know what nerves supply the recti; and for want of knowing this, we may be wrong in our diagnosis of the seat of disease in the central nervous system.

Our pathology should be both particular and general. We should try to make it as wide in its principles as our physiology will let us, but at the same time as precise and accurate as our anatomical knowledge will permit. Just as we study, as physiologists and anatomists, the vegetative life of general tissues, the structure of organs for special functions, and the universal harmony of most diverse functions in individuals, so we ought, as workers in the field of practical medicine, to study every case that comes before us; as presenting:—

(1) Disease of Tissue. (Changes in tissue.)

(2) Damage of Organs.

(3) Disorder of Function.

Learn the tissue health of a patient, as shown by the colour of his hair, the elasticity of his skin, the tint of his complexion, the feel of his arteries, and the condition of his urine. Examine the chief organs, and with especial care the one in fault, or supposed to be in fault; and

lastly, be most careful to ascertain if organs near or distant to the one or more most damaged act in harmony —if function be disordered. Of course, disease of tissue damages the organ of which it forms part, and disorders its function, but that special tissue may be diseased or liable to it in nearly every other part of the body, and again the damaged organ may disorder the functions of most distant parts.

My lecture will be chiefly a series of recapitulations of the above general views by special instances. I prefer to take as my first illustration a common general disease which produces much local damage to several organs, viz., rheumatic fever. This disease is exceedingly common at this Hospital, and as a consequence, valvular disease of the heart and chorea are also common, and now and then as a further consequence, embolism. I take this illustration in preference to one of disease of the nervous system, as a method, to have any real root in nature, should apply to diseases and diseased conditions generally and not to a limited nosological group.

Rheumatic fever is a blood disease, in which the fibrous tissues are attacked. The pericardium and the endocardium suffer, and the valves of the heart especially suffer as they contain a good deal of this tissue. Next, as a consequence of the disease of fibrous tissue in these valves, the heart (the organ) is damaged as the segments of its valves do not fit, and thirdly, as the heart is the central organ of the circulatory system, the functions of all other parts, but especially those of the lungs and liver, are liable to be disordered. Now, notwithstanding the importance of the organ affected, we have in rheumatic fever to think more of inflamed fibrous tissue than of heart disease; and the question as we stand by the bedside ready to prescribe, is how to prescribe for a patient who has rheumatic inflammation of his fibrous tissues, and just then the information as to the precise valve affected is comparatively insignificant.

I am aware that some would treat inflamed fibrous tissue in the valves of the heart with greater energy than they would similar disease in less important parts, as it is desirable at even great risk to get rid of lymph effused in fibrous tissue so importantly situated. It is just as a general in war will defend a point, not on account of its size or intrinsic importance, but on account of its strategetical value. The strategetical value of the flaps of fibrous tissue (the mitral valves), between the left auricle and left ventricle is very great.

Suppose the turmoil of tissue changes is over, and that the patient has got "well," as he supposes, and perhaps even as we thought too. A year later, he comes to us again, and we find that he is suffering from disease of the heart. We must now think most seriously as to the kind of damage to the organ. For, although the damage is exceedingly *local*, it is in an essential part of the mechanism of a most *central* organ. We get to know generally the kind of damage by auscultation and by percussion. Still we cannot treat a damaged organ as a surgeon can; he can make an artificial pupil, or nail two bones together, but when tissue changes are over, we can do little to reverse them. We must pass next to the disorder of function. The patient often does not know that he has disease of the heart, but comes because he has dropsy or breathlessness. He does not come for his damaged organ, but for disorder of function consequent on derangement of its mechanism. We may find a bruit when a patient attends for some other disease, but we can do nothing for that. We can never make the valves fit, nor remove vegetations from them; so that it is of more moment to study the functions disordered, which we can help, than the organ damaged which we cannot repair. But information about the latter is of great value, as it helps us to gain a clear idea of the former.

Here let me give you a piece of advice. Never treat a patient on stethoscopic evidence only. The heart may be a bad musical instrument and yet a good force-pump.

However noisy the first sound may be at the base of the heart, if the apex be in its right place, if there be no signs of hypertrophy, no anæmia, if the pulse be good, and, above all, if the patient does his work well, we have nothing to treat. To treat such a case would be to treat a sound. It would be just as great a mistake as the drunken captain made, who tried to navigate his vessel round a speck of dirt on his chart, which he mistook for an island.[1] And in cases of aortic or mitral regurgitation, we could only at the most give such remedies as would improve the general health, or possibly act on the muscular tissue of the heart itself to enable it to keep up the stream of blood in spite of the imperfection of its valves. But we should render the patient much more service by advising him how a man with a damaged heart should choose his occupation, so that the circulation of important organs might not be obstructed, or in other words, that function might not be disordered.

The best illustration I can give you of disorder of function in heart disease is the pneumonia, so-called, of mitral obstruction. It is frequently the immediate cause of death in heart disease. But see how different this disorder of function is from real pneumonia, in which the tissues of the lung are affected. In one the lung's business is obstructed, in the other its texture is diseased. The congestion of the first is because the blood cannot get out of the lungs, from obstruction to the column at the mitral orifice; of the other, because it is held there by the tissues. And after the stage of congestion, there is still a perceptible difference in the progress, the first tending to so-called apoplexy of the lung and hæmoptysis, and the other to effusion of lymph into the air-cells, and to hæmoptysis too, but of a different kind.

[1] "Generally speaking, I would say that the tendency of half-instructed auscultators is to over-estimate the importance of the murmur *as a fact*, and to under-estimate it *as a means of investigation*; to pay too much attention to the mere existence of the sound, and too little to the circumstances in which it occurs."—Gairdner, *Clinical Medicine*, p. 561.

Fatty degeneration of the heart, the hypertrophy of the left ventricle, found so often in chronic Bright's disease, and disease of the aortic valves, say from hard work, as hammering, are all "diseases of the heart." But, as I hope to show you when I speak of diseases of this organ, in their clinical association with diseases of the nervous system, the significance of each for diagnosis and treatment is very different to that of the others. In all the same organ is damaged, but they are diseases of different tissues, and, as I think, produce different symptoms, *i.e.*, disorder differently the functions of other parts or organs.

In the eye, in the organisation of which most of the tissues of the body are used, we may find plenty of illustrations of the method I have mentioned. And here I may say that the study of disease of tissues may be carried on with greater advantage in the eye than in any other part of the body. We can watch the changes in a nodule of lymph on the iris, and can see the retinal degeneration attending chronic Bright's disease. Here only lies a nerve termination, with an artery and a vein, under our view.

We should all study diseases of the eye if we wish to know diseases of the nervous system, and indeed pathology generally. Besides the importance of this knowledge as a help to the study of the physiology of disease (for six of the nine cranial nerves have to do directly or indirectly with the organ of vision), it is a field for the study of diseases of tissues. It is, I think, of quite as much importance as a practical knowledge of the chemistry of the urine. In passing we may remark that it is quite erroneous to suppose that the introduction of the stethoscope, ophthalmoscope, and laryngoscope has made our medical lives more laborious. On the contrary, we have more things to do, but we do our work more easily, and far more satisfactorily.

Now, in the eye, it is of infinitely more importance

to recognise the early stage of disease of tissue—the stage of change—than to learn the exact part of the organ of vision damaged. But I admit that we often can in eye diseases, by the ophthalmoscope, detect both the tissue changes and the part of the organ where they are, and often by empirical experience tell the constitutional cause of the disease. It is, however, of importance to study diseases of the eye for other purposes, even when the active changes are over, for their value as showing the particular tissues undergoing change. Thus, syphilitic choroiditis and syphilitic inflammation of the pia mater —the choroid of the brain—are pretty much alike as diseases of tissue, although they produce very unlike physiological symptoms, as the organs they damage, and the functions disordered are very different. If I may use such expressions in pathology, the "idea" is the same, but the "events" are different. See the practical bearing of this. A patient comes to us with acute pain limited to a spot on one side of his head, and partial "amaurosis." Now, if we find the "amaurosis" to depend on syphilitic choroiditis, the information is most valuable, however old the choroid disease may be, and however hopeless the treatment of that might now be. For it would give us good grounds for hope that the acute limited pain on one side of the head was the sign of a new syphilitic inflammation of a similar tissue, the pia mater, and that our treatment in the stage of change might save the brain from damage, although it might do no good to the older disease of the choroid which (like mitral valves, damaged by a bygone attack of rheumatism) had passed through the stage of *change*, in which we may help, to one of permanence, the effused products having, in fact, become a bastard tissue, and only in degree less vital than the legitimate tissues.

I will take a few more illustrations from the eye. Choroiditis and retinal apoplexy are alike as damages of organs, but different as diseases of tissue. Presbyopia

and paralysis of the ciliary muscle are pretty much alike as disorders of function, but very different as diseases of tissue. Thus, the latter is due to disease of the third nerve or to that part of it which supplies the ciliary muscle, whilst the former is chiefly a change (in the firmness of the lens) of advancing years. And it is well to remember this as a fact, and I mention it to you as I was once puzzled because I did not think of it. A patient seventy years of age had paralysis of the third nerve on one side, but the sight on the paralysed side was quite as good as on the other. The reason of this apparent exception was that the accommodation of both eyes had failed long before, and paralysis of the ciliary muscle could not make them worse. He was already completely presbyopic, so that really instead of saying the eyes were equally good, we should say in such a case that they were equally bad.

In an organ like the ear about which medical men, or physicians at least, generally know little, it is obviously of more importance to ascertain that the patient is suffering arthritically or syphilitically, than to get to know what is the exact part of the organ which is damaged.

In diseases of nerve trunks, see how exceedingly different are the physiological symptoms (the disorders of function) of disease of the optic, the portio dura and the ulnar nerves, although they may be quite, or closely, alike as diseases of tissue. A nerve trunk besides its nerve tubes consists of fibrous and connective tissue. It is compound, and in a loose sense is an organ. Suppose it damaged by syphilis, as in syphilitic neuroma. Here you see the important fact to be known is, that the patient has syphilitic disease of fibrous and connective tissue, an exact knowledge of the individual nerve-bundle attacked and the functions disordered being of secondary importance.

The transition from nerve-trunks to the central nervous system is easy. Never forget that every organ is made up of constituents which exist more or less in the greater part of the body. We have in nervous masses,

nerve-cells and fibres, blood-vessels, and also connective tissue. So, too, we find that "Diseases of the Nervous System" are not always diseases of nervous tissue; but often of other tissues which enter into the composition of nervous organs—of blood-vessels, connective tissue, &c. Now, it is a well-known fact, that syphilitic deposits are generally found on the surface of the brain. They begin in the pia mater and *extend* into the nervous mass; in short, they affect the connective tissue where it is in bulk. There is, although this is denied too, connective tissue in the substance of the brain itself, but I never saw deposits unconnected with the pia mater; the reason being probably the simple one that there is very little, if any, connective tissue in the central masses of the nervous system.

It may seem contradictory to say that I have no experience of syphilitic diseases of nervous matter, when in the next breath I speak of syphilitic epilepsy and syphilitic paralysis. But you will readily see that it is really important to know whether the epilepsy be due (directly or indirectly) to disease of the connective tissue in or on the nervous masses or to some change in the nerve-fibres or nerve-cells themselves. We cannot set to work to treat an *organ* like the corpus striatum, but we may prescribe for a patient who has syphilitic disease of connective tissue, wherever it may be. Now syphilitic deposit in the pia mater is essentially the same as a node of the tibia, or a cellular node of the skin, or of the spleen and liver, and it requires the same treatment as these. I cannot too much impress on you that we ought above all things, in brain disease to study diseases of tissues. It is of infinitely more importance than to know the exact organ of the brain damaged; for instance, to know that there is lymph effused in the pia mater, than that the attendant epilepsy, the disorder of function of part of the brain, has certain physiological peculiarities. Indeed, for my part, I fear we are too late, for cure at

least, when the epilepsy is well established. Then, the tissue changes in the pia mater have settled down, the brain is damaged, and its functions will continue to be disordered more or less in spite of what we can do. It is in the stage of inflammation, when, as I have before said, the patient comes with pain on one side of his head that we may help, not when he is "well" of this and a few months afterwards comes for epileptiform seizures affecting the opposite side of the body. It is when the pia mater is in a condition analogous to acute iritis and not in one analogous to occluded pupil from bygone inflammation of the iris.

Now for still more illustrations of these principles, and I hope I am also teaching you some of the details of brain disease in giving them. You may, perhaps, say that you cannot help, instinctively, carrying on the three trains of thought in investigating a case. Now I deny this. A little observation will tell you it is incorrect. Do we not sometimes treat convulsions by specifics, with too little thought as to the exact part of the brain damaged and as to the real tissue diseased, and, more broadly, do we always take the condition of the patient into equal consideration in thinking of his disease? Indeed, the paroxysm, the disorder of functions, is often taken for the whole disease when it is only the outward part of it by which we ought to try to learn the rest. It is not at all a rare thing to hear of a patient, after his first attack of epilepsy, being treated as if the convulsions were a sign of inflammation—disordered function being taken as evidence of tissue changes, of inflammation or congestion. Now, after a severe fit of epilepsy, and especially after a series of fits, there is a kind of congestion, but it is no more related to inflammation than the pneumonia of heart disease is to true pneumonia. There is too much blood in the vessels of the brain; but it is there because it cannot get along, and not because it is held by the tissues. Here then let me impress on you not to take the

most violent symptoms of disordered function of the brain for inflammation, and remember this especially in injuries to the head. In children it is notorious that convulsions frequently signify just the reverse—debility. A rigor, which may attend suppuration anywhere, is far more valuable information that tissue changes are taking place in the head than convulsions. Now remark, I here give no opinion as to whether you should bleed or not in either the condition of congestion of disordered function, or in the genuine congestion of inflammation. All I say is this, know what you are about. What you do, do advisedly, and do not bleed by routine for "head symptoms," because the organ damaged is an important one.

To think of a case as one of convulsions, or as epilepsy, without reference to the tissue changes and the organ really in fault, is a very careless way of thinking. The epilepsy of a young man coming on gradually, and persisting for years, is generally quite a different disease to that of a man of middle life with rigid arteries and signs of degeneration, although the convulsion, the disordered function, may be quite alike. In a case like this, how very foolish we should look if we had diagnosed brain disease. A young man supposed to be quite healthy, falls down in convulsions. He soon becomes sensible again, but his urine is found to be bloody and albuminous, and it is ascertained that several of his brothers and sisters are then suffering from scarlet fever. Here, obviously, the disease was disorder of function of the brain, and the organ most anatomically changed was one remote —the kidney.

I have seen a patient treated for delirium as if it were due to "congestion" of the brain, with no benefit, who was sent to sleep at once by brandy and water. It was common at one time to treat mania as if it were due to inflammation of the brain; but in the last case I saw, although the patient was furious, bottled stout and brandy were given, and with benefit.

How very different is true croup which occurs in a healthy, perhaps in a robust, child, and which impedes the entrance of air by disease in the larynx, from false croup, which comes on in a ricketty child, and impedes respiration by partially closing the glottis. (By the way, false croup is generally called "fits" by the nurse.) One is disease of the tissues of the organ, and the other disorder of a mechanical function. Here the treatment is obviously quite different. Then, too, how often has disorder of function of the larynx, from pressure on the recurrent laryngeal by a tumour in the thorax, been taken for disease of the larynx itself.

Let me repeat then for the last time, before my final illustrations, that in each case we must study changes in tissues, examine the organs damaged, and investigate the disorders of function. But remark, although inseparable, each of the three trains of thought ought to be distinct, or the more information we get the more confused we shall be. Let me illustrate this. In a cerebral case you examine the eyes, and record the following facts: The patient has an arcus senilis, a large pupil, paralysis of the sixth nerve, white atrophy of the optic nerve, and the retinal degeneration of Bright's disease. Now, it might be a nice arrangement to put all these things down under the heading of "eye symptoms," and, indeed, as an index, might be useful. But the student who thought of these different symptoms from one point in his own mind would have learnt many things, but would after all know little. He might be very minute, but would never be precise. He would attend to the superficial contiguity of events, and neglect the more real continuity of ideas. Such a man's classifications and arrangements would have the merit of neatness, but he himself would have a most unmethodical mind. There would be a superficial appearance of order, but a real incoherence. I think this kind of order is the very worst form of slovenliness. For some of these eye symptoms are merely local

signs of almost universal changes. Let us never forget than an elaborate arrangement is often the highly-worked cloak that covers up inferiority of organisation. The most gorgeous piece of mosaic is not equal to the simplest natural flower. The mere minuteness that lacks precision is always ready to deal in generalities, but never arrives at a scientific generalisation. Of what value would be our minute inquiries as to convergent strabismus if we did not distinguish whether it was due to paralysis of the external rectus or to hypermetropia? Indeed, the more minute a man is who does not think of his anatomy, or who studies merely one side of disease, the more harm will he be likely to do to the science of practical medicine.

I take hemiplegia as the last illustration, and, for ease of description, hemiplegia on the left side. Hemiplegia is the simplest, the best known and the commonest disease of the nervous system.

(1) A patient may have hemiplegia from syphilitic disease of the pia mater on the surface of the right hemisphere.[1]

(2) A patient may have hemiplegia from embolism of the right middle cerebral artery.

(3) Because blood has been effused in the right corpus striatum.

(4) Because a tumour has grown in, or grown into, the right corpus striatum.

Now, in all these cases, the hemiplegia would be of the same kind. We might choose four cases in which the physiological symptoms would be alike, even in degree, to a nicety. For the organ damaged, the corpus striatum, would be, so far as the hemiplegia is concerned, just the same.

[1] Although the obvious disease is found here, there can be little doubt that the hemiplegia is due to some secondary change in the motor tract. There is no fact better established clinically, than that very extensive disease of the hemisphere may exist without any hemiplegia. I shall, in a future communication, give my reasons for believing that disease on the surface of one hemisphere, when it does cause hemiplegia, does so by producing disordered circulation in an arterial region—that of the middle cerebral artery. This vessel supplies the corpus striatum, as well as the part (the hemisphere) on the surface of which is the most obvious disease.

But to think of these cases baldly under the name of hemiplegia, as an entity, would leave us with very vague grounds for treatment. We should be on a par with the public, who ask what is good for a cough? In the first case, we should have to treat syphilis; in the second, limited softening of the brain; in the third, some more general affection, of which degeneration of arteries was the most prominent feature, and rupture of one or more vessels in the corpus striatum the most dramatic event; and in the last, tumour, if we knew how. In all, we should treat the patient.

If, then, a patient comes to us with hemiplegia, we study him as well as his local disease—his tissue changes and general health, as well as his paralysed arm and leg. If he be past fifty, if he have rigid arteries, an arcus senilis, if he have Bright's disease, and above all, if the attack were sudden (although it might not be suddenly complete), we diagnose effusion of blood in nervous tissue. But if he be a young man having healthy tissues, and if the attack began in his usual health, we ought to see if he have not valvular disease of the heart, and ask if he has had rheumatism or scarlet fever. If it began slowly, or if it followed an attack of unilateral convulsions, and especially if preceded by pain in the opposite side of the head, we should seek for evidence of syphilis. (Here I may just say that the clinical history of the epilepsy and paralysis from syphilis is like that of the same diseases which follow injury to one side of the head, in this, that the convulsions and the paralysis are generally quite unilateral.) To recapitulate: (1) syphilitic deposit on the surface of the hemisphere; (2) softening from plugging of an artery; (3) blood effused in nervous tissue; and (4) a new growth.

Having given you instances of damages of the same organ by different affections of its tissues, I will now give you instances of damages of very different organs, the result of similar tissue changes. The following four cases

of hemiplegia are each of a different and well-marked kind from disease of the same sort, but at different points in the motor tract :—

(1) A patient has suddenly paralysis of the left arm and leg, and of a very small part of the face, and slight deviation of the tongue. Clot in the right corpus striatum.

(2) Paralysis of the third nerve on the left side, and of the arm and leg and of a small part of the face on the right. Clot in the left crus cerebri.

(3) Paralysis of the portio dura, and complete deafness on the left side, paralysis of the arm and leg on the right. Clot in the floor of the fourth ventricle involving the nuclei of the left facial and auditory nerves and also the fibres which have just crossed coming from the limbs on the right side.

(4) Sudden and complete paralysis of the tongue, palate, and of the vocal cords all on the left side, weakness of the left limbs, and slightly of the right. Clot, involving the lingual and spinal accessory nuclei, and implicating the fibres from the limbs, about their decussation.

Now, how exceedingly various are the sets of symptoms produced by the same tissue changes. But we have to think, as regards treatment, of effusion of blood in nervous tissue, rather than of disease of the corpus striatum, crus cerebri, pons varolii, &c. To attend to the seat of the damage only is to study merely the physiology of disease, and is not practical medicine.

I should be misunderstood if I were supposed to underrate the physiological study of diseases of the nervous system. Indeed, I think that to neglect it shows want of education, but to neglect the clinical shows want of experience and sagacity. Never forget that we may run the risk of being over-educated and yet under-cultivated. We may be gorged with facts in anatomy, physiology, and pathology, and yet our faculties may be little trained for investigating disease. Sometimes the physiological history and the clinical history point in the same direc-

tion. Thus, paralysis of part of the face, with slight defect of articulation, shows central disease; and this information, with evidence of wide tissue degeneration, points to the conclusion that our patient has not only a degenerated nervous system, but that it is now breaking up. Again, a patient (the last of the four cases just given) has paralysis of the palate and of the tongue, and of the internal muscles of the larynx on one side. This is physiological evidence that he has disease, as Lockhart Clarke's researches clearly show, at the nuclei of the lingual and spinal accessory nerves. This, then, shows that the disease is in a dangerous position, for although it may be local it is very central, and, besides, perilously near the so-called vital knot. So much for the physiological history of the case; and now for the clinical. The patient has albuminous urine, but no dropsy. He has, too, the degeneration of the retina found so often in chronic Bright's disease, or, as it might be vaguely called, "partial amaurosis." So, then, he has already *central* disease, in the medulla oblongata, and he has, too, signs of wide degeneration of *general* tissues, which render him liable to further damage in other parts of the nervous system.

In future lectures we shall study individual cases more closely.

TETANUS DOLOROSUS, AND THE RELATION OF TETANUS TOXIN TO THE SENSORY NERVES AND THE SPINAL GANGLIA.

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THE evidence obtained by Gumprecht (3), Courmont and Doyon (2), and others, of the carriage of tetanus toxin by way of the peripheral nerve trunks to the central nervous system, independently of the paths of blood and lymph, has been abundantly confirmed by the recent researches of Meyer and Ransom (8). These authors have shown that tetanus toxin circulating in the blood or in the cerebro-spinal fluid has apparently no immediate action upon the cells of the central nervous system. The neurones appear to be protected from the poison with a very remarkable efficiency by the thin walls of the blood and lymph spaces, and to be attacked only by toxin which reaches them by the long path of their axis-cylinder processes, after its absorption at the motor nerve endings in the muscles. It is apparently only at these nerve endings that the circulating poison can come into sufficiently intimate contact with the neurone protoplasm, and it is the motor nerve-fibres which provide the unique mode of access of the toxin to an undamaged central nervous system. The greater part of the so-called "incubation" period is taken up by this relatively slow carriage of the toxin from the periphery, and is roughly proportionate to the distance in different animals between periphery and centre.

Meyer and Ransom show, however, that, with the toxin in circulation, a local violence done to the nervous system, as by a puncture made into the spinal cord, which will bring

toxin into direct relation to the neurones, rapidly produces, without this "incubation" period, the characteristic symptoms of tetanus poisoning, localised in the neurones about the site of injury. Similarly, in the undamaged animal, precisely localised tetanus may be produced by direct injection of the toxin to definite parts of the spinal cord or to the peripheral nerves, and here, apparently, the necessary condition of success is such a disturbance of the structures as will bring toxin into actual contact with the neurone or with its axis-cylinder process.

Quite apart from their special pathological importance, and their significance in relation to the mode of action of the the antitoxin, these results raise questions of considerable neurological interest. In the astonishingly local character of its action, due, apparently, to an extremely low power of diffusion, tetanus toxin differs from all the well-known poisons which act on the nervous system. Meyer and Ransom have expressed their belief that the toxin is conveyed to the neurone body, there to display its action, by a cellulipetal streaming along the axis-cylinder process, and at the same time their experiments give no indication of a transference of the toxin from one neurone to another, or from one cell group to another. If the suggestions arising from their work be confirmed, the action of this poison may find a new importance for the study, not only of the trophic relations of one distant part and another of the same neurone, but also of the chemical as well as of the anatomical relations of one neurone and its processes to another within the nervous system.

In the course of some experiments Professor Meyer has been good enough to allow me to make in his laboratory, some results were obtained of neurological rather than of pharmacological interest, and these may be shortly noted here. They refer to the interesting form of sensory tetanus recently described for the first time by Meyer and Ransom under the name of "tetanus dolorosus." I am greatly indebted to Professor Meyer, not only for his hospitable kindness, but also for the whole technique of the toxin manipulations involved in the experiments. These were

carried out entirely in the Pharmacological Institute in Marburg.

I.—The Relation of the Spinal Ganglion to Tetanus Dolorosus.

The poisoning by tetanus toxin of the sensory apparatus, like that of the motor, is expressed in signs of hyperactivity. It is not obtainable by injection of the toxin to the blood or the lymph, or even into the substance of a peripheral sensory nerve. It is only to be observed after injection of toxin into a posterior root—when it may be obtained in a pure form—or after injection directly into the substance of the spinal cord, which generally produces a mixed sensory and motor tetanus. With toxin circulating in the blood, a rapid onset of mixed motor and sensory tetanus follows so slight a disturbance of the cord as the insertion of a needle and the injection of a $\frac{1}{2}$-drop of an indifferent salt solution (Meyer and Ransom, p. 385), and both the motor tetanus and the hyperalgesia correspond segmentally with the region of the disturbance. Here the indifferent injection, presumably by destruction of capillary walls, effects the direct access of the toxin from the blood or lymph to the neurones, and is equivalent, indeed, to an injection of toxin. Whether or not the sensory nerve endings, like the motor, are able to take up toxin from the circulation or the lymph spaces has not been determined, but at all events toxin brought by direct injection to the fibres of a peripheral sensory nerve does not produce tetanus dolorosus. This immunity of the peripheral sensory nerves will be noticed in Section II. below.

The remarkable symptoms of tetanus dolorosus, which have been described in detail by Meyer and Ransom, appeared to offer an opportunity for gaining a comparison between the motor neurone of the anterior horn and the sensory neurone of the posterior root and the spinal ganglion. Hitherto these two neurones have been considered as being in a sense correspondent. Head (4), in discussing the homology of herpes zoster with acute anterior poliomyelitis, goes so far as to say that "the posterior root

ganglion is the exact equivalent of the anterior horn. For whereas on the motor side the cells of the neurons of the lowest level lie in the anterior horn, on the afferent side they happen to lie collected together outside the spinal cord in the posterior root ganglion." It might be expected then, on such a view as this, that just as after its injection to an anterior root the toxin, having passed cellulipetally along their axons to the anterior horn cells, produces strictly localised symptoms of motor tetanus, so the localised hyperalgesia, produced by injection of toxin to a posterior root, is due to a cellulipetal passage of toxin to the corresponding spinal ganglion, and to a resulting increase within the ganglion cells of their receptive or discharging functions. This is the comparison I have tried to institute, but it will be seen that it is not supported by the experimental results obtained.

The following experiment provides an example of the striking positive evidence which may be obtained of the absence of any share taken by the spinal ganglion cells in the production of tetanus dolorosus :—

Experiment I.—July 15.—Young dog (2½ months). Weight, 5,650 grammes.

July 15.—5 to 6.30 p.m.—Paraldehyde narcosis (1·3 cc. per kilo). A.C.E. later. Lumbar posterior roots, 5, 6, 7, prepared on the right side. Tetanus toxin injected to posterior root 6 (126,000 + ms. = 22 + ms. per gramme).

July 16.—Dog lively and well. No symptoms of tetanus.

July 17.—Same. No symptoms.

5 p.m.—Injection repeated into the same root. (A.C.E. narcosis.) (180,000 + ms. = 31 + ms. per gramme.)

July 18.—8 a.m. No symptoms. Dog well, and has good appetite.

9.30 a.m.—Hyperæsthesia began in the right gluteal region, and at the root of the tail on the right side. From this time onwards typical tetanus dolorosus became more and more pronounced. The dog, on the slightest touch upon the affected gluteal area, turned defensively and bit at this region. Spontaneous symptoms began to increase, the dog referring discomfort or pain to the area from time to time without external stimulus. The impression received from the animal was that hyperæsthesia

gradually passed to hyperalgesia, the localised discharges of painful impulses becoming more and more easily provoked, until they had the appearance of spontaneity. There was no sign of motor tetanus or of any increase of the ordinary tactile-motor reflexes.

11.15 a.m.—A.C.E. narcosis. The lumbar posterior roots 5, 6, 7, were divided between cord and ganglia.

12 a.m.—On waking from narcosis "spontaneous" attacks of sensory tetanus reappeared. These occurred at intervals of fifteen to sixty seconds, becoming more frequent as time passed. The sensations were always referred to the original gluteal area, the dog turning round to the right as before to examine the apparent site of pain, and usually to gnaw or bite at it. Attacks of this kind were now no more easily elicited by stimulus of the gluteal area than by that of any other part. The animal had previously lain only on its left side; it now lay on the affected right side, which it had never done since the first hyperæsthesia began.

It was clear that the spinal ganglion of the injected root, and those of the roots next above and below, had no share in the sensory tetanus.

12.50 p.m.—A.C.E. narcosis. The spinal cord was divided below lumbar 7, and the lumbar posterior roots 3, 4, 5, 6, 7 (together with the anterior roots, except in two cases), were cut on both sides.

2 p.m.—The hind quarters and limbs paralysed and anæsthetic. As the narcosis failed, the periodic attacks began again. In exactly the same way they occurred at intervals, now, perhaps, more regularly, about once in three or four minutes. Just as before, the sensations were very obviously referred to the original gluteal area. Between the attacks the dog remained quiescent and somnolent. No trace of motor tetanus.

3 p.m.—Death given by HCN.

Post mortem.—All lumbar roots from third to seventh inclusive, both anterior and posterior, on both sides, found cut, except anterior roots 5 and 6 on the right side. Below the seventh root the whole cord found divided. No bleeding found, and no signs of cord compression. The dura was undamaged, except at the hinder section.

The first point to be noticed in this experiment is the failure of the first injection of toxin. The injection needle happened no doubt, to be so inserted as to bring the poison

only within the lymph spaces of the endoneurium without disturbing the nerve-fibres sufficiently to give the toxin access to the axis cylinders. The difficulty of securing intoxication of the actual fibres even by injection among the nerve bundles stands in striking contrast with the ease with which other nerve poisons may be effectively applied to medullated nerve-fibres. The possibility of tetanus toxin existing in the connective tissue spaces of a nerve without specific action upon the fibres, is a point which will be referred to again below, in Section III.

Fourteen hours after the successful second injection, the first hyperæsthesia was noticed. During the earlier stages of the sensory tetanus it is only by stimulus of the hyperæsthetic area (which first appears as a small hyperæsthetic point) that the typical response may be obtained. The animal gives no more than natural attention to stimulus of any other part of the skin, but the touch upon the affected area makes the dog turn at once to defend itself from the cause of stimulus. Later, the response may be obtained by touching or moving other parts of the body, or even without apparent stimulus from outside, but in every case the sensation is referred with precision, as the behaviour of the dog clearly indicates, to the original area of sensory tetanus corresponding with the posterior root taken for injection.

The fact that the sensory tetanus continues unchanged after complete severance from the cord of the ganglia of the injected root, and indeed of all the adjacent ganglia on both sides, is conclusive evidence that the spinal ganglion has no share in the production of tetanus dolorosus. The results are hostile to the view that the spinal ganglion cells have centripetal discharging functions which may be intensified in tetanus poisoning, and, if the assumption commonly made be adopted, that the motor tetanus is an expression of an increased discharging activity of the anterior horn cells under the influence of the toxin, then the posterior root no longer appears to be "the precise equivalent of the anterior horn."

It is to be noticed that the passage of the toxin injected to the posterior root is not cellulipetal, as in the anterior

root, but cellulifugal, while both are centripetal. The localised hyperæsthesia must be taken as a sign of increased excitability or increased activity, not of the spinal ganglion cells, but of cells within the cord, or, conceivably, of the central cut ends of the posterior root fibres themselves. There is no indication on other grounds that tetanus toxin has an exciting action upon axon processes independently of its action upon the neurone body; that, and the length of incubation period in sensory tetanus, make it unlikely that the poisoned cut ends of the posterior root fibres are responsible for the maintenance of tetanus dolorosus after severance from the ganglia. This is a point, however, which is to be settled by further experiment. If, as seems probable, the localised hyperalgesia has its seat in poisoned cells of the gray matter, we have here the first evidence of a transference of the toxin from one neurone to another. Such a transference at this point would be particularly interesting in view of the observations of Anderson (1), to be referred to again below, which indicate an intimate trophic relationship between the centrally connected fibres of the posterior root and the cells in the cord.

II.—The Immunity of the Peripheral Sensory Fibres.

It has been shown by Meyer and Ransom (p. 389) that the sensory nervous system enjoys a remarkable immunity from tetanus poisoning. Injection of the toxin to the blood, or subcutaneously, or into a peripheral nerve trunk, never produces tetanus dolorosus. The sensory tetanus can only be set up by access of the poison to the spinal cord substance, or at least to the posterior root fibres; that is to say, it must be introduced to the nervous system central of the spinal ganglion. The authors conclude that the spinal ganglion cell acts as an "insuperable obstacle" to the further passage of toxin coming centralwards after its absorption by the peripheral fibre. If, as in the motor fibres, the toxin is conveyed centripetally by a streaming of the axis-cylinder protoplasm, it must be supposed to pass

into the cell and to remain there, or leave it, without any contamination of the centrally-directed neurone process.

The striking fact that injection of toxin to the blood gives a general but purely motor tetanus and injection to the sciatic trunk a localised but again a purely motor tetanus might be explained on the supposition that the motor nerve-fibres possess a greater affinity for the toxin than the sensory fibres, and, in a mixed nerve, take up from the latter the toxin which has reached them. Meyer and Ransom have described one case of injection to a pure sensory nerve—the N. infraorbitalis (*loc. cit.*, p. 398). The result was not quite clear; no symptoms of hyperalgesia, no tetanus dolorosus arose, unless a frequent sneezing which was noticed is to be taken in that sense. After ten days a slight motor tetanus, localised in the ear muscles of one side, appeared, due, perhaps, to the superficial petrosal connection with the facial nucleus.

I have tested this question of peripheral sensory immunity by cutting the anterior roots of the lumbar plexus and giving time for degeneration, so that toxin might be injected to the sciatic nerve containing only sensory axis cylinders. An example is given in the following experiment :—

Experiment II.—July 22.—Cat, 2,020 grammes.

4 p.m.—Paraldehyde (1 cc. per kilo). A.C.E. later.

Right anterior roots lumbar, 3, 4, 5, 6, 7, and sacral 1, were cut and the cauda equina divided behind. Wound dressed with iodoform gauze and closed.

July 23.—Complete paralysis of the right leg and the tail. Sensibility normal. Left leg normal. Cat eats well and has no sign of fever.

July 24.—The same. Wound dressed. Good appetite.

July 25.—The same.

July 26.—The same.

July 27.—The same.

5 p.m.—A.C.E. narcosis. Right sciatic exposed high up in the thigh, and tetanus toxin injected into several different bundles. (Total dose, 1·3 million + ms., = 640 + ms. per grm. wt.)

July 28.—As before. Cat healthy and eats well. No sign of tetanus dolorosus.

6 p.m.—A.C.E. narcosis. Toxin injected to lumbar posterior root 7 (430 + ms. per grm.)

July 29.—8 a.m.—Hyperæsthesia began and typical tetanus dolorosus rapidly established. No trace of motor tetanus.

The animal was destroyed.

Post mortem.—On the right side. Lumbar anterior roots 4, 5, 6, 7, and sacral 1, were all cut and showed degeneration. Lumbar posterior roots 4, 5, 6, 7, and sacral 1, were all normal. Lumbar posterior root 7 was the root of injection (distinguished by a silk thread).

On both sides, all roots behind sacral 1 were cut in cauda equina. All roots in front of this on left normal.

Preparations made from some of the roots and ganglia, to be referred to below.

This experiment supplies strong, if only negative, evidence of the completeness of the immunity to tetanus poisoning possessed by the sensory fibres, even in the absence of contiguous motor fibres. A similar experiment in the case of a dog may be quoted.

Experiment III.—July 20.—Young hound (1 year), 1,280 grammes.

5 p.m.—Paraldehyde (1·3 cc. per kilo.). A.C.E. later.

Spinal roots in right lumbar region prepared. Anterior roots lumbar 4, 5, 6, 7, and sacral 1, cut outside the dura. Cauda equina divided below. Wound dressed with iodoform gauze and closed.

July 21.—10 a.m.—Dog rests quietly on the right side. Right hind leg and the tail paralysed without loss of sensation. Defæcation and micturition efficient.

4 p.m.—The dog is lively, eats well, and has no signs of discomfort or fever.

July 22.—As before. The dog walks about freely on three legs and eats well.

July 23.—10 a.m.—A.C.E. narcosis. Right sciatic exposed, and tetanus toxin injected into several nerve bundles (2·2 million + ms. = 172 + ms. per grm.). The dose given was accidentally larger than intended.

4 p.m.—Antitoxin (80 million-ms.) subcutaneously injected to the right shoulder.

July 24.—The dog well and lively. Eats well. Wound dressed daily and gives no discomfort.

July 25.—Same. Dog walks about on three legs. The right hind leg remains normally sensitive.

1.30 a.m.—A.C.E. narcosis. Toxin again injected to the right sciatic high up in the thigh (1·5 million + ms. = 117 + ms. per grm.).

July 26.—No change. Dog is lively and healthy.

July 27.—No change. A slight œdema in dependent right ankle.

6 p.m.—A.C.E. narcosis. Tetanus toxin injected into the right lumbar posterior roots (70 + ms. per grm.). Wound reclosed. It was clear in this injection that the small amount of toxin injected (0·1 cc.) passed only to the perineural sheath.

July 28.—10 a.m.—No symptoms of tetanus. Animal healthy and well.

6 p.m.—Injection repeated to same root (70 + ms. per grm. again).

July 29.—8.30 a.m.—Typical tetanus dolorosus established, rapidly becoming more intense. The animal was destroyed.

Post mortem.—On right side. Anterior roots lumbar, 4, 5, 6, 7, and sacral 1, found cut, and all show microscopic degeneration of ten to twelve days.

Posterior roots lumbar 4, 5, 6, 7, and sacral 1, all normal, and show no degeneration.

On left side, all roots to sacral 1 (inclusive), normal.

On both sides, all behind sacral 1, cut and degenerated.

It will be seen that too large a dose was given by accident in the first injection. Meyer and Ransom find the minimum lethal dose of tetanus toxin for the dog to be 20 to 25 + ms. per grm. body weight, by subcutaneous injection, or 4 + ms. per grm. by injection to the sciatic. The corresponding figures for the cat are respectively 200 + ms. and about 100 + ms. per grm. body weight. The first injection in Experiment III. above was 172 + ms. to the partly degenerated sciatic. It was feared that of this amount a lethal dose might be expected to pass to the lymph or blood, and accordingly, after six hours, a large dose of antitoxin was given to remove the excess of toxin in circulation. Such an injection of antitoxin would not after that delay effect the neutralisation of toxin already contained within the axis-cylinders or travelling along them (Meyer and Ransom and others). As no

results followed the first injection, another large dose was given two days later. This was nearly thirty times the minimal lethal dose for the intact sciatic, and was injected like the former between the fibres of several of the nerve bundles. Assuming that the injected toxin did not pass entirely to the endoneural spaces in this case, but had access to the nerve-fibres, a condition relatively easy to secure in the case of the sciatic, and assuming that the rate of passage of the toxin along the axis-cylinders (if there be such a passage at all in the case of the sensory fibres) is the same as along those of the motor nerves or the posterior roots, then the poison, in the absence of any delay or block at the spinal ganglion, should have reached the central nervous system at latest within twenty-four hours. No symptoms of poisoning appeared, however, in the three days following this large injected dose.

Owing to operative difficulties, it is less easy in the case of the posterior root to secure a successful injection; and, indeed, the first attempt in this experiment failed, the tiny mass of injection passing obviously to the connective tissue sheath. The injection, however, on the 28th, into the posterior root, produced typical tetanus dolorosus in fourteen hours.

The evidence supplied by the experiments described is, from the nature of the case, negative, but, so far as it goes, it strongly supports the contention of Meyer and Ransom that no sensory tetanus can result from the poisoning of the peripheral sensory nerves, either by direct injection or by way of sensory nerve endings after subcutaneous injection. These authors assume a passage of the toxin along the poisoned sensory fibres, like that along the motor axis-cylinders, and imagine a block to its further effective course to be offered in some way by the cells of the posterior root ganglion. At present such a block is entirely conjectural, and, indeed, there is no experimental ground for the assumption that the peripheral sensory fibres can either take up the poison or transmit it.

There is a large body of evidence which indicates a fundamental difference between the peripheral and the

central process of the sensory ganglion cell. The comparative morphology of the cell and its two processes, as Retzius (11) has pointed out, makes it probable that the central process, originally reaching the centre after a long course, is the homologue of the axis-cylinder process of other nerve-cells, while the peripheral process, originally passing after a short course to the more peripherally placed ganglion, is to be compared with the dendron processes of other cells. Now at present we have only evidence of the absorption of tetanus toxin by, and its passage along, the motor axis-cylinder processes. We have no evidence of its passage along dendron processes, and, indeed, it is possible that the astonishing localisation of the poison in particular cells and cell groups within the central nervous system, after localised injection, is a mark of the inability of the dendron processes to further this passage.

The pathology of tabes offers further evidence of important trophic differences between the central and peripheral axons, and this evidence is confirmed by the well-known observations of Lugaro (6), who found that section of the posterior root produced little, while section of the peripheral process produced very considerable, degenerative changes in the spinal ganglion cells. Anderson (1) has extended these results by examining the condition of the posterior root fibres after early section of the root. He found that this section checked the development of the portions of the fibres left still attached to the spinal ganglion, without checking the development of the spinal ganglion, or of the peripheral afferent processes. He has suggested that degeneration of the cut root fibres on the ganglion side occurred because normally "the chemico-physical equilibrium in these processes is maintained not only by the spinal ganglion cells, but also by the intraspinal cells with which these processes are anatomically continuous" (p. 512). It is possible that the tetanus toxin has no action upon the peripheral sensory neurone as such, and that in tetanus dolorosus, as in motor tetanus, only intraspinal cells are poisoned. Anderson's demonstration of a trophic relation between the posterior root fibres and intraspinal

neurones may provide an explanation of the effective centripetal carriage of injected toxin along the posterior root fibres to the intraspinal neurones.

Unluckily, it is not easy to define the relation of the toxin to the peripheral sensory nerves by finding positive evidence of the movement of the poison along the axis-cyclinders in one direction or another. It is impossible to exclude the entry of toxin to the lymphatics of the nerve trunk, or to decide, when the presence of poison has been determined in a given segment of nerve, whether it has been contained within the axis-cylinders or only within the lymph passages. This difficulty is illustrated in the next section.

III.—The Conveyance of Toxin in the Lymphatics of the Nerve Trunks.

Meyer and Ransom have asserted that the poison in the motor axis-cylinders moves *only* in a centripetal direction, but they have given no evidence of the absence of a simultaneous centrifugal movement, and no evidence of a movement of the toxin without extension, that is, of its desertion of one part of the neurone and its transference to another. Twenty-four hours after injection of toxin to the sciatic, they found that three pieces of the nerve, one taken at the site of injection, the others respectively central and peripheral of it, were all equally poisonous to mice inoculated with their emulsions. They believed, with probability, but without exclusion of participation of the axis cyclinders in the result, that the toxin in this case had been conveyed up and down the nerve by the lymphatic channels described by Key and Retzius (5), within the connective tissue sheaths of the nerve bundles.

In an experiment like one of those given above with a "sensory" sciatic, I hoped to be able to gain evidence of some movement of toxin in the sensory fibres, and perhaps, if the block supposed by Meyer and Ransom to occur at the ganglion be actual in fact, to find an accumulation of toxin in the spinal ganglion and its absence from the posterior root. The results obtained however, though interesting from

another point of view, allowed no such determination to be made. These are shortly given here.

Experiment IV.—In Experiment II. above, tetanus toxin was injected to the right lumbar seventh posterior root (after an innocuous injection to the "sensory" right sciatic). Typical localised sensory tetanus, confined to a defined gluteal area, followed. The animal was killed, and some of the blood centrifugalised. Emulsions were made in measured quantities of normal salt solution of a piece of the right sciatic high up in the thigh, of the right spinal ganglia, lumbar 4, 5, 6, and sacral 1 (the seventh lumbar being omitted as the level of injection) and of the right fifth lumbar posterior root between ganglion and cord. Control emulsions were made in the same way of the corresponding structures on the left side. The small pieces of nerve were separately rinsed from superficial blood contamination before being crushed into emulsions.

Small measured quantities of these emulsions and of the serum were injected to mice subcutaneously. The results appear in the following table:—

Mouse weight	Material inoculated	Weight	30th	31st		1st		2nd		3rd		4th	
			5 p.m.	9 a.m.	5 p.m.	9 a.m.	5 p.m.	9 a.m.	5 p.m.	9 a.m.	5 p.m.	9 a.m.	5 p.m.
(*a*) 8 gr.	Blood serum	·5 gr.	Inoc.	O	O	=	≡	≡	≡	+			
(*b*) 26 gr.	R. sciatic nerve	·035 gr.	,,	—	≡	+							
(*c*) 23 gr.	R. spinal ganglia L. 4, 5, 6 and S. 1	·034 gr.	,,	O	—?	—	—	=	=	≡	+		
(*d*) 23 gr.	R. post. root L. 5	·012 gr.	,,	O	—?	=	=	≡	+				
(*e*) 25 gr.	L. sciatic nerve	·035 gr.	,,	O	O	—??	—??	O	O	O	O	O	O
(*f*) 22 gr.	L. spinal ganglia L. 4, 5, 6 and S. 1	·036 gr.	,,	O	—	=	≡	+					
(*g*) 23 gr.	L. post. root L. 5	·01 gr.	,,	O	—?	—	=	=	≡	≡	=	=	=

O No symptoms. = Marked local tetanus.
— slight local tetanus. ≡ General tetanus.
+ Dead.

It will be seen that the blood was poisonous, but the amount injected was relatively so large per body weight that it is difficult to compare its toxic value with the others obtained, though it is clear that this value was too small to vitiate by contamination the other results. The most striking feature of the table is the abundant presence of toxin, not only in the spinal ganglia on the right side, which it may have reached from the injected sciatic, but also in the fifth posterior root (the seventh only having been injected) on the right, and in the ganglia and fifth root on the left uninjected side. The left sciatic contained no toxin, or only the smallest trace.

Here we have positive evidence of the passage of toxin, not only to the uninjected fifth root on the right side, but also across to the posterior root and the ganglia of the opposite side. Apparently no poison had gone so far as to descend along the left sciatic. Since there was no trace of any sensory tetanus on the left side, it may be concluded that the toxin on this side at least was contained within the lymph channels, and had found no entry to the axis-cylinder processes.

This result makes it quite clear that the ordinary inoculation method of testing for the presence of toxin in a given piece of nerve allows of no discrimination between toxin circulating, freely, and perhaps, for the time being, harmlessly, and that, on the other hand, effectively contained within the axis cylinders. By another method it is hoped that the true "intra-neural" course of the toxin can soon be traced, and the ambiguous results of inoculation just described are now mentioned only to be set against some other recent observations of the same kind.

Morax and Marie (9), in the laboratory of M. Roux, have carried out an examination of a large number of the nerves of a horse after death by intramuscular injection of tetanus toxin. The nerves were tested for the presence of toxin by the inoculation of their emulsions into mice. The original toxin injection was made into the left gastrocnemius of the horse, and the animal succumbed in a little less than three days. The subsequent tests showed that not only the blood

and cerebrospinal fluid were poisonous, but also the emulsions made from a very large number of nerves. The left sciatic was naturally very poisonous; the right sciatic markedly, but less poisonous. The lumbar anterior and posterior roots on both sides were strongly toxic, and about equally so. Strikingly more toxic than these, however, were the cervical sympathetic, the masseter nerve, the vagus, and the lingual. The substance of the spinal cord in the lumbar region only gave local tetanus on inoculation (except the left pyramidal bundle, which gave death in four days); in the dorsal region it contained no toxin, or only a trace, and the substance of the bulb was quite innocuous. The optic nerve was free from toxin.

On the ground of these experiments the authors conclude that "the three types of peripheral neurones, the motor, the sensory, and the sympathetic, are equally able to absorb the tetanus toxin."

It is quite clear that no such conclusion as this is justified by the described observations. The inoculation tests allowed of no differentiation between toxin in the neurones and that in the endoneural lymph spaces, and my own results already given indicate an extreme probability that the larger part, if not the whole, of the toxin in an infected nerve may be present only in the lymph channels without entry to the neurone processes.

It has been seen how unlikely it is that the sensory neurone, or at least its peripheral process, takes up the toxin equally with the motor neurone. With regard to the neurones of the visceral system no comparison can yet be made. Meyer and Ransom, by injection of toxin to the vagus in the dog, have obtained a slowing of the pulse lasting for a week or more, without other symptoms, and ending in complete recovery. Morax and Marie, as we have seen, claim that the sympathetic neurones "take a very conspicuous part in the absorption of the tetanin," but this rests only on the inoculation result, and is not supported by the description of any symptoms of tetanic hyperactivity of the sympathetic supply to the head. These authors suggest that the vaso-motor fibres absorb the poison by way of their nerve endings in the vessel walls.

It may prove later to be possible to effect the production of tetanus poisoning of the sympathetic neurones, but, so far, experiment shows it to be incomparably more difficult to infect these neurones than those of the somatic system. In the rabbit and the cat I have soaked the superior cervical ganglion and the sympathetic trunk in the neck with a strong solution of the poison for half an hour or more, and have made punctures through the solution into the ganglion or nerve (a thin india-rubber sheet protecting the tissues below) without obtaining any symptoms of poisoning or of any increased activity of the sympathetic neurones. On this question clinical evidence throws little or no light at present.

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NOTES ON THE MINUTE STRUCTURE OF THE HUMAN CAUDATE NUCLEUS AND OPTIC THALAMUS.

BY JOHN TURNER, M.B.ABERD.

I HAVE recently attempted to stain pieces of the caudate nucleus and optic thalamus by my methylene blue and peroxide of hydrogen method with successful results in a few cases.

As these are regions about whose structure very little, and about whose functions less, is known, a description of my results may be of interest, even if only to compare with those obtained by Golgi's method. A feature which strikes one forcibly is the very great resemblance, nay, absolute identity of appearance, between some of the cells shown by my method, and certain of the cells in different parts of the central nervous system shown by Golgi's method. The appearances recorded in both the caudate nucleus and optic thalamus refer to adult cases, and the tissues were taken from the brains of the insane (in the case of the caudate nucleus transversely across the broad end of this body where it abuts on the lateral ventricle).

CAUDATE NUCLEUS.

Sometimes crowds of darkly-stained cells are shown scattered about without any attempt at definite orientation. The great majority of these are of much the same size (averaging 13·5 μ by 11 μ, roughly speaking), but occasionally one very much larger is seen (27 μ by 18 μ). They are of various shapes—square, pentagonal, triangular, pyriform, &c. By careful inspection under a high magnification the axons can frequently be detected, and they leave the cell in a peculiar

manner: a long (8 μ to 14 μ), somewhat sausage-shaped or triangular mass projects from the cell body, and is stained less deeply than it; down the centre of this, or along one side, the dark fine thread of the axon can be seen passing to the cell body. I have not yet seen this mode of origin of the

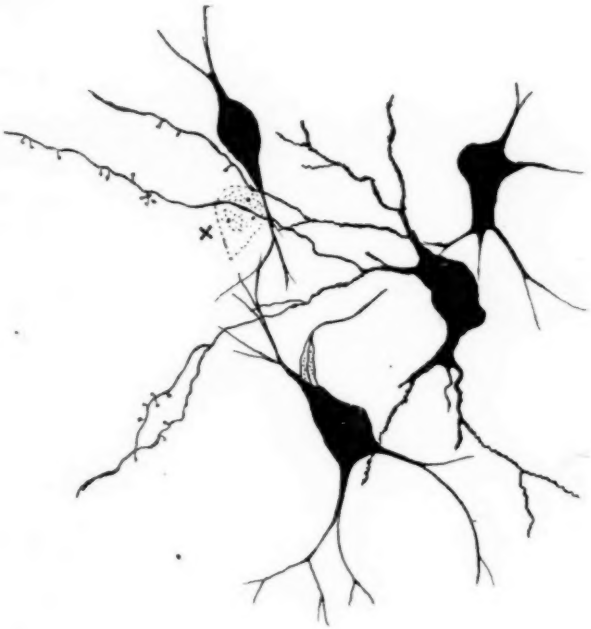

FIG. 1.

Caudate nucleus. Shows a group of five cells. One (X) is very faintly stained, and with a granular nucleus. Three have short, rapidly thinning, rectilinear dendrites, and in the lower one the axon is represented. The remaining cell has very long sinuous dendrites, with lateral appendices.

axon in any other part of the central nervous system. Although at first sight these dark cells appear very similar, yet, on looking carefully at them, it can be seen that there are distinguishing features by which some can be separated from the others. They all give off processes from their

different angles, but in some cases these rapidly dwindle in calibre: they divide near the cell body; each branch thus formed again divides a little further on; and now the divisions are so fine and pale that they disappear from view (see fig. 1). Throughout their short course these branches are rectilinear. Other of the cells give off fairly numerous darkly-stained dendrites, which do not divide in the regular dichotomous manner of the first, but have a more sinuous course, and can be followed for very long distances without very decided diminution in calibre, and the more distal portions of which are fairly thickly beset with little lateral projections terminating in a swelling (figs. 1 and 2). In some cases, whilst this latter class of cell stains out very decidedly, the former apparently proves refractory, and does not stain (see x in figs. 1 and 2), or very faintly, appearing as pale blue cells with a round, well-defined, granular nucleus. I say apparently, because I am not yet able to definitely assert that they are the same as the darkly-stained cells with short dentrites previously described, although I believe them to be so.

In the cases where the dark cells with long branches alone stain out distinctly, their dendrites form a coarse felt work of comparatively stout branches (*i.e.*, coarse in comparison to the intercellular plexus shown in the brain cortex by my method), spreading about in all directions, and even with comparatively low magnification (x300), the lateral projections are clearly visible. I have not yet seen any structure approaching in appearance to an investing network over any of the caudate nucleus cells.

A. Hill gives illustrations of cells in the lenticular nucleus of hedgehogs, stained by Golgi's method, which appear in most of their characters to be identical with the dark cells, having long dendrites which I have just described, except that he states that he was able occasionally to trace a fine fibril beyond the little knob or swelling, or occasionally several beads arranged linearly, but without a visible connecting thread. I have not been able in my cases to trace any continuous structure beyond the swelling on the lateral processes.

FIG. 2

Caudate nucleus. Darkly stained cell with long sinuous dendrites beset with lateral appendices. Some at **A** rather longer than others. The long fibril **B** seems to spring from a dendrite, but I cannot be certain on this point. The other fine rectilinear fibres are probably axons or collaterals. **X**=a very pale cell with granular nucleus.

S. Soukhanoff and T. Geier figure cells stained by Golgi's method in the posterior horn of the spinal cord of kittens, rabbits, and human beings, which also appear identically the same as these caudate cells with long branches. T. Geier finds that in new-born animals the lateral appendices invest the whole dendrite, and often appear on the cell body, whilst, apparently, as the animal matures, they disappear more and more from the cell, and the proximal parts of the dendrites, only continuing at the more distal parts. In my cases in the great majority of instances the lateral appendices are only situated on the dendrites at some distance from the cell body (see fig. 2). S. Soukhanoff figures from an old guinea-pig a posterior horn cell which is closely beset with lateral appendices all over its body.

I believe that the cells in the caudate nucleus with long dendrites and numerous lateral appendices are the homologues of the similar dark cells met with in the cerebral cortex, but here the lateral spines do not terminate at the first little swellings seen not far from the main dendrite, but fibrils pass off from them which ultimately form the pericellular network.

There is a very striking resemblance between the structure of the caudate nucleus and some parts of the frontal cortex. We meet in both cases with apparently similar dark cells, but those of the frontal region give off finer dendrites, whose lateral appendices are more delicate, and therefore not such conspicuous objects, although they are more numerous here, and in many cases it is possible to trace beaded fibrils which spring from these dendrites, passing after a longer or shorter course directly into the pericellular network.

The development of the central nervous system shows that the cortex and caudate nucleus have a similar origin, and are probably of similar nature. Possibly the dark cells of the latter region, with lateral appendices, are the forerunners, so to speak, of the dark cortical cells with long lateral beaded fibrils, the one being a further development of the other.

From the point of view which I have taken in my

previous communications on the subject, viz., that the dark cells (of the cortex) represent a mechanism for the conduction and distribution of afferent impressions to the networks over the pyramidal cells, the appearance of similar cells in the posterior horns of the spinal cord is very interesting. Apparently these cells do not occur in the anterior horns. The large multipolar cells here, which in all probability have a motor function, or which at least are concerned with efferent impulses, do not show the lateral appendices.

The nerve-cells do not show excess of pigment or the peculiar loculated condition which are common features in those of the optic thalamus. They are often invested by numerous round nuclei. Spider cells are very inconspicuous and small, whereas in the optic thalamus in most cases they are large and very numerous.

Optic Thalamus.

Most of my sections were taken transversely through the pulvinar, and through the anterior tubercle; a few were taken antero-posteriorly from above downwards to include in one piece both the anterior tubercle and the pulvinar.

There are certain very striking points of difference between the cells of this body and those of the caudate nucleus; they are much larger (21 μ by 18 μ), their dendrites are smooth, and never, so far as my observations go, show lateral appendices. On the other hand, there are some similarities. There is the same tendency for the cells to divide themselves into two groups, pale and dark; but, as with the caudate nucleus, so here this tendency is not nearly so well marked as in the case of the cerebral or cerebellar cortex. In successful preparations one group always stains darkly, whereas the other may at one time be only very faintly coloured, and at another may be nearly or quite as dark as the former. When, however, all the cells are darkly stained there are other features, as was the case in the caudate nucleus, by which those of one group can be distinguished from those of the other.

I will describe first of all those cells which sometimes

stain very faintly and at others very deeply, and for this purpose it is advantageous to make use of some that have stained darkly. Perhaps the commonest shape is square or pentagonal; from each angle a stout smooth dendrite passes off in a straight course. These branches divide near the cell body, rapidly dwindle down to very fine threads, and are lost to view (see fig. 3). When these cells are only

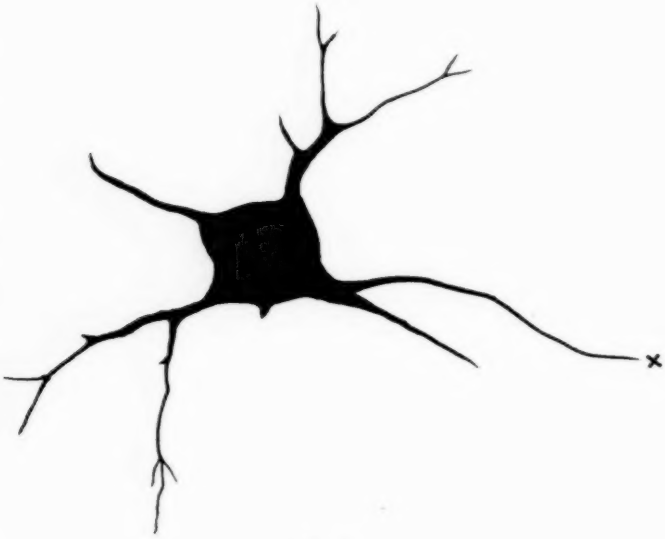

FIG. 3.

Optic thalamus. A cell with short rectilinear dendrites. x probably represents the axon.

faintly coloured (I am assuming that they both belong to the same group, but on this point I wish to make the same reservation as with the caudate nucleus cells) their nucleus is large, clearly defined, and sparsely granular, with one or two granules considerably larger than the others, but in the dark condition they appear homogeneous. Sometimes one dendrite with a neighbouring portion of the cell body is

very darkly stained, whilst the remaining part is very pale; such an appearance lends support to the view that the above-described cells belong to the same group.

The other variety of cell, that which in successful preparations always stains darkly, is of much the same size and shape as those previously described. The dendrites can be traced for long distances, running a somewhat sinuous course, and branching pretty frequently. Although the branches are smooth, *i.e.*, devoid of lateral appendices, they are generally varicose. Apparently in a normal condition they retain a fairly stout calibre at a very considerable distance from the cell body (fig. 4), but in a case of general paralysis, the optic thalamus of which I examined, they rapidly became very attenuated, although they could still be traced just as far in the form of very slender threads, which bore here and there varicosites dark in outline and with colourless centre (ring-like). There does not seem to be any attempt at a definite orientation of the cells.

I have not yet seen any structure resembling a peri-cellular network, but Turner and Hunter state that they found it in the case of certain animals by the vital method of staining.

The above-described cells are fairly regularly distributed throughout the pulvinar and within the anterior tubercle. In places here and there one encounters small clusters of rather large oval, pyriform, or bipolar cells, crowded together in the form of cell nests. As I have not succeeded in getting these to react properly to the stain, I cannot give any further detailed description of their characters. In some places adjacent to the anterior tubercle there are large tracts which do not show any decided nerve-cells, but a collection of nuclei, some of which pertain to small spider cells, others are apparently free, and others, perhaps, belong to nervous elements where the body has not stained. Some of these nuclei stain much deeper than others, but they are all of nearly the same size.

Quite a prominent feature in most of the thalamic sections is the wealth of big spider cells, which send out numerous rather short, wavy, not very sharply defined

branches (see fig. 4). In the sections examined from a fairly advanced case of general paralysis I did not see any of these elements, but so far this has been the only one in which they were not to be found.

A fairly large number of axons can be seen coursing about the sections in different directions. They follow a rectilinear course, and stain very faintly, except in short

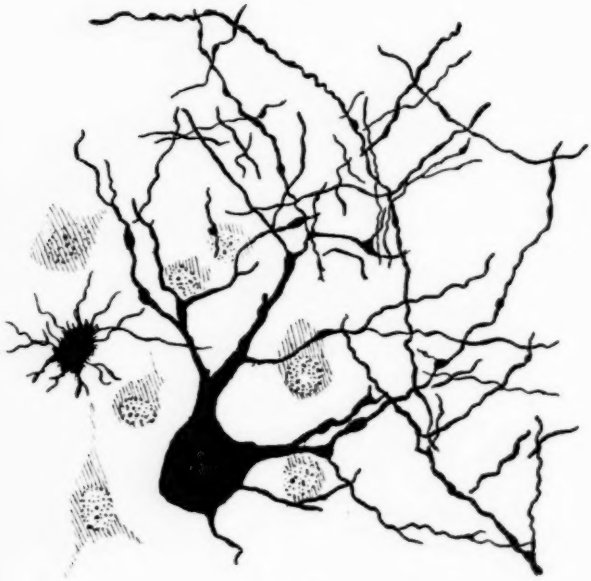

FIG. 4.

Optic thalamus. A dark cell with long, freely branching, varicose dendrites. Dendrites of other cells whose bodies are not in the figure are shown, forming a coarse felt work. Seven cells with faintly stained bodies and granular nuclei can be seen, and a large spider cell.

areas here and there, which probably correspond to a node of Ranvier. On account of their small size and pale colour they are very inconspicuous objects. Pigment is a marked feature in most of the cells. Fig. 4 gives a very good idea of the general appearance of the structure of the optic

thalamus in successful preparations. It shows a dark cell and the somewhat coarse felt work which the varicose dendrites form. Seven pale cells with granular nuclei are shown, and to the left of the picture a large spider cell.

Serge Soukhanoff and T. Geier have made a special study of the appearances presented by the cornual cells in human and animal spinal cords by Golgi's method, and they show that the dendrites of the anterior horn cells are longer, smoother, more rectilinear in their course, and rarely possess lateral appendices, although they frequently are varicose. The dendrites of the cells in the posterior horns, on the other hand, are shorter, ramify more freely, follow a tortuous course, and are plentifully beset with lateral appendices, which may be short, fine threads ending in a knob, or more complicated and longer filaments (*rejetons* of Soukhanoff).

The similarity in all their aspects of these posterior horn cells and those of the caudate nucleus with long dendrites, which I have described, appears to me to afford us sufficient justification for considering them of the same or of a closely allied nature. And as the posterior horn cells are generally allowed to be chiefly concerned with the passage and distribution of afferent impulses, so I would submit that the caudate cells, with similar morphological characters, are of a like nature physiologically.

Soukhanoff, to account for the differences between the anterior and posterior horn cells, supposes that dendrites rich in lateral appendices belong to cells which appear as nerve units, less subordinate to other neurones, and which have a directing (*directrice*) function. He compares them to the other cells which by Golgi's method show numerous lateral appendices, viz., the pyramidal cells of the cerebral cortex, and the Purkinje cells of the cerebellum, and would differentiate them from cells of Golgi, Martinotti, and Cajal.

The anterior horn cells he regards as having a more subordinate *rôle*, depending for their activity on the cortical cells of the cerebrum, and executing the orders of these latter, and also depending on the reflex excitations from the first sensory neurones.

I have elsewhere called attention to the fact that whilst

my method very clearly shows lateral appendices on the dendrites of Purkinje cells, it never reveals the slightest trace of such on the pyramidal cells of the cerebrum, and I can now speak after examination of many hundreds of sections from the brains of seventy to eighty different adult human beings. I do not believe that lateral appendices exist on the dendrites of the pyramidal cells; that they are shown by Golgi's method I admit, but I believe that the structures which this method reveals are not a part of the dendrites on which they appear, but belong to the beaded network which I have shown surrounds for a very long distance the dendrites and apices of these cells.

Further, by my method Martinotti cells stain deeply, and present the characters of the other dark cells, the dendrites of which *do* show a quantity of very delicate lateral filaments.

For these reasons, and because the pyramidal and Purkinje cells stain faintly, whilst the Martinotti cells stain deeply, I classify the former apart from the latter, with which I include the other dark cells of the cortex and those in other regions. I believe that the marked distinction in the staining properties of the two groups represents a more fundamental distinction than the morphological differences represented by the presence or absence of collateral appendices. Nevertheless, we frequently meet with cells usually belonging to the pale class which take on more or less dark colour. It is well to bear in mind Erb's dictum that nature is not schematic, and does not recognise hard and fast boundaries. We cannot, as it were, pigeonhole one class of cells into a compartment quite distinct from another.

Whilst I believe on other than morphological grounds alone that the cells of Purkinje, in spite of their undoubted lateral appendices, are allied to the pyramidal cells, inasmuch as both are concerned with the conduction of centrifugal impulses, I am inclined to look upon the dark cells of the optic thalamus, in spite of the absence of lateral appendices, as allied to the dark cells elsewhere. It is quite possible that the reason why they are absent here has to do

with a relatively much simpler connection between the thalamic cells than between the cells of the cortex. I would suggest also that the relatively rudimentary condition of the lateral appendices of the caudate nucleus cells is due to a similar reason.

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Note.

All the figures were outlined with the aid of a Zeiss Camera Lucida under the same magnification ($\frac{1}{12}$ oil immersion Beck and No. 2 eye-piece).

Figs. 1 and 4 have been slightly reduced in reproduction.

HEREDITARY SPASTIC PARAPLEGIA. ITS RELATION TO FRIEDREICH'S DISEASE AND ITS CLAIM TO BE CONSIDERED AS A CLINICAL ENTITY.

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HEREDITARY spastic paraplegia is the term under which cases have from time to time been described which in many ways closely resemble those described by Friedreich, especially in their tendency to affect several members of a family, but on the other hand they differ sharply from the Friedreich group in the fact that their predominant features are those of a lateral rather than a posterior sclerosis. In spite of this and other differences these cases have hitherto only usually been alluded to in connection with irregular cases of Friedreich's disease, and have not yet been generally treated as a separate division which they would seem to deserve. The sporadic cases, which, as I shall show later, occur with considerable frequency, appear to have still more completely shared the fate of their family group, and to have been passed over altogether, or to have been again looked upon as occasional variations of Friedreich's disease which did not admit of further classification.

In this paper I propose to show that hereditary spastic paraplegia occurs both in families and in isolated instances; that the isolated cases are by no means uncommon, and that the two together form a distinct clinical group, which, while belonging to the broad group of cases which fall under the term of hereditary ataxic paraplegia, can nevertheless be

clearly differentiated from Friedreich's disease, Maries group of hereditary cerebellar ataxy, and other conditions which they may at first sight seem to resemble.

Since Friedreich first established the entity of the disease that bears his name there have been many opinions expressed as to whether such definite boundaries can be recognised as were originally laid down. Passing over the broader question as to whether the disease can be separated from locomotor ataxy and disseminated sclerosis, a question which has long ago been settled in the affirmative, there still remain a number of cases among which are those already alluded to, which, although resembling Friedreich's disease in many ways, differ from it sufficiently in others to make the question of their inclusion doubtful. Before referring to my own cases, which I believe to be sporadic examples of hereditary spastic paraplegia, it will be advisable to clear the ground by mentioning the symptoms that are usually accepted as making up a typical case of Friedreich's disease, and by referring also quite briefly to some of the variations that have been met with.

Ladame,[1] in a well-known article to which writers frequently refer, sums up the symptoms of a typical case of Friedreich's disease as follows: Slow and progressive ataxy of the four limbs—usually attacking several children of the same family, dating often from very early age—commencing in the legs, extending gradually to the trunk and arms, the muscles of the larynx, those of the tongue and eyes; weakness of legs, increasingly difficult gait, tabeto-cerebellar, (Charcot); choreiform unsteadiness; static ataxy (Friedreich), difficulty of articulation; nystagmus; spinal curvature; paralytic club foot; abolition of knee reflexes; no sensory disturbances; absence of oculo-pupillary anomalies and of lightning pains; integrity of sphincters.

In the last edition of Quain's "Dictionary of Medicine," the symptoms of Friedreich's disease as laid down by Bastian for the purpose of differential diagnosis are as follows:—

Onset commonly before the twentieth year; affections of

[1] Brain, 1890.

sensibility generally absent; no pains; double vision, and Argyll-Robertson pupil absent; nystagmus frequent, often late and slight; oscillations of head and trunk very common; knee-jerks usually absent and no clonus; rigidities rare; difficulties in speech; tremors of arms exaggerated by movements; gait reeling and unsteady, of cerebellar type; no mental disturbances; pes cavus and lateral curvature of the spine.

Of course it must be recognised that certain minor variations will be sure to occur occasionally, especially in respect to the absence of one or more symptoms, but it is with regard to the more pronounced differences that questions of classification have arisen.

Sanger Brown[1] was one of the first to propose to extend the symptomology of the disease in order to include his well-known cases in the group. Thus, in comparing the symptoms of his cases with those of Freidreich's disease as laid down in Sir William Gowers' text-book, he says: "If the age at the time of onset were considerably advanced, and if it were stated that the knee-jerks and skin reflexes might be greatly exaggerated, that in some cases there might be ankle clonus, and that there might be optic atrophy, the cases herein reported would be fairly well provided for."

Sanger Brown's proposal to classify his cases in this way was adversely criticised in the same number of BRAIN as the original communication appeared, by Ormerod and Bernhardt of Berlin. Neither of these authors was able to agree to the inclusion of the cases under the head of Friedreich's disease, but Bernhardt admits that if we drop the appellation of Friedreich's disease, and are satisfied with such a name as hereditary, familial, or generic ataxy, then the cases would come under this category. The way was, however, eventually further cleared by the separation of a definite group by Marie,[2] which he described under the title of "hérédo-ataxie cerebelleuse," and it is in this group that most writers now think that Sanger Brown's cases should be included.

The chief characteristics upon which Marie relied for the

[1] BRAIN, 1892, vol. xv. [2] *La Semaine Médicale*, 1893.

identification of his cases from those described by Friedreich were their later age of onset, exaggeration of deep reflexes, the frequent presence of optic atrophy, and the absence of spinal curvature, deformities of the feet and other trophic changes.

As scoliosis and club foot are so common in Friedreich's disease, and as I shall have occasion to allude to these points more particularly later on in speaking of my own cases and their classification, it will be as well, perhaps, to quote Marie's own words regarding these points. He says: —"L'absence de troubles trophiques dans l'hérédo-ataxie cérébelleuse mérite d'être relevée, car c'est à l'état de pure exception que l'on voit survenir chez un malade de Klippel et Durante (Francois H.) la chute des ongles, chez un malade de Sanger Brown (Case 5) un certain degré d'amyotrophie, chez d'autre malades du même auteur la diminution du poids de corps. Le point important c'est que dans l'hérédo-ataxie cérébelleuse on ne trouve (à part peut-être pour le cas de Botkine dans lequel est notée une légère cyphoscoliose) aucune trace de la scoliose si fréquente dans la maladie de Friedreich typique, non plus que du pied bot spécial à cette dernière affection et qui s'y montre d'une façon tellement précose qu'il en constitue, dans certain familles, l'un des meilleurs signes de début."

In order to make the differences that are now generally accepted for teaching purposes between the two groups quite clear, I will give the following table from "Church and Peterson's Text Book on Nervous and Mental Diseases."

Friedreich's Form.	*Marie's Form.*
Hereditary spinal ataxia appears usually before puberty.	Hereditary cerebellar ataxia appears usually after puberty.
Choreic movements in upper extremities and oscillations in head and trunk frequent.	Very common and pronounced.
Optic atrophy and amblyopia very exceptional.	Common.
Tendon reflexes diminished or absent.	Increased and foot clonus frequent.
Club foot and scoliosis common.	Exceptional.

Marie's cerebellar group, then, being separated off, it now remains to examine the so-called irregular cases that are still unclassified. Sir William Gowers[1] in alluding to the subject says that variations appear to be more common in isolated cases where the disease affects an only child or only one member of a family, and, in speaking of these isolated cases, says: ". . . Many of these cases, however, differ somewhat, although sufficiently characteristic in their main features to justify their inclusion . . ."; and he particularly mentions variations connected with speech, micturition, and tremor.

It is in connection with these irregular cases that Sir William Gowers mentions the cases of family spastic paraplegia recorded by Howard Tooth, and in finally summing up the relations between Friedreich's disease, the cerebellar cases and the family spastic cases, says:—

"It might already seem as if we had a series to deal with, with Friedreich's ataxy at one end and family spastic paralysis at the other, the intermediate space being occupied by cases like those of Sanger Brown, in which spasticity and ataxy are combined, and in which the underlying anatomical conditions as regards the cord is one of sclerosis affecting lateral and posterior tracts fairly equally, while in Friedreich's disease we have to deal with a condition in which the posterior columns suffer most, and in family spastic paralysis with one in which the lateral columns may be exclusively affected."

The family cases of spastic paraplegia alluded to above are those described by Dr. Howard Tooth.[2] The predominant feature of these cases, of which four were recorded, was that of lateral sclerosis. The knee jerks were exaggerated in all of them, and in three there was also deformity of the feet. The family tendency of the disease was shown by the fact that the four cases only represented two families.

The epitome given by Tooth of his cases is as follows:—

Case 1.—Onset at 15 years of age, with weakness of back, clonic spasms of right leg, with stiffness of both legs, stammering

[1] "Diseases of the Nervous System," vol. i., 3rd Edition, Gowers and Taylor.

[2] *St. Bartholomew's Hospital Reports*, vol. xxvii., 1891.

speech same time, lately becoming inebriate in character, with tendency to giggle. Strong extensor rigidity in both legs. Exaggerated knee jerks. No pain. No loss of sensation. No eye symptoms. No ataxia. Slight incontinence ot urine.

Case 2.—Brother of last case. Onset at 15 years of age, after an accident, with stammering speech. One year after, cramps, weakness and stiffness of legs, mostly right. No weakness of back. Great rigidity of legs. Keee jerks greatly exaggerated. Pes cavus. No loss of sensation. No pains. No eye symptoms. No ataxia. No incontinence.

Case 3.—Backward child. Did not speak or walk till 2 years of age. Onset at 9½ years of age with shuffling gait. Later spastic gait. Strong rigidity, extensor and adductor, both legs. Exaggerated knee jerks. Speech slow. Dribbles at mouth. Tendency to uncontrollable laughter. No eye symptoms. No loss of appetite. No pains. No ataxia. Pes equinovarus. Slight weakness of sphincters.

Case 4.—Elder brother of last case. Scarlet fever at 2½ years of age. Deafness and discharge from ears ever since. From that time has had shuffling gait. Six years speech stuttering. Slight extensor rigidity of right leg. Knee jerk exaggerated. Slight clonus. Genu valgum. No loss of sensation. No pain. No ataxia. Slight weakness of sphincters.

In commenting on his cases Tooth says: "We are driven, then, to the conclusion that we have here a true primary lateral sclerosis, comparable with the hereditary posterior sclerosis first described by Friedreich. In this connection it should be remembered that Friedreich's disease may, in some rare cases, present the superadded signs of lateral sclerosis."

Dr. Tooth also refers to the paper by Dr. Gee in which three cases of spastic paraplegia occurring in the same family are recorded, and thinks that his own cases, taken together with these, should go towards establishing the claim of this affection to be considered as a separate type or variety.

Weston D. Bayley[1] has described a family in which five generations at least have presented cases of primary lateral sclerosis.

He gives notes of nine cases, all of which with one

[1] *Journal of Mental and Nervous Dis.*, November, 1897.

exception date their condition as beginning between the third and the seventh year.

There was no ataxia, deformity of the feet was common, and the knee-jerks were exaggerated in all the cases except one. Bayley says: "The group of symptoms presented by these cases clearly places the lesion as a primary lateral sclerosis of the crossed pyramidal tracts. The wonderful similarity of the cases both as to the time of mode of onset, absence of sensory phenomena, and (excepting the first case) state of reflexes, established its hereditary type. It is obvious, therefore, that another congenital affection must be placed in the lists with the already well-known Friedreich's ataxia and pseudo-hypertrophic paralysis." Bayley points out that it differs from these two diseases and also from the spastic paraplegia of infancy, which "is cerebral in origin, involves the upper extremities with the lower, presents cerebral symptoms, and is believed to be absolutely non-hereditary." Cases have also been described by others, among whom Church and Peterson, refer to Strümpell, Bernhardt, South, Newmark, Achard, and Fresson.

It seems clear, then, that there is a group of cases analogous to those described by Friedreich in their tendency to run in families, and, this being the case, in order to make the set complete one would expect also to find isolated cases where only one member of a family suffers, as experience shows, frequently occurs in the case of Friedrich's disease.

It is the existence of such isolated cases that I shall now attempt to show from the cases which I have myself met with.

The first case is that of a young man aged 21, who was sent to my out-patient department by Mr. Aslett Baldwin, whom he consulted on account of his foot. For five years, *i.e.*, since the age of sixteen, he had been troubled by his toes sticking up, and it was for that he now sought relief.

In the left foot the four outer toes were in the hammer position, but the big toe was almost normal. In the right foot the toes had also begun to assume the same position, but not to such a degree as in the left. There was not any definite pes cavus. Both knee jerks were increased, but the left more so

than the right. The plantar reflexes were brisk but flexor in type. There was no nystagmus, no alteration of speech, no loss of sensation. There was slight incoördination, which could be demonstrated by making him try to walk along a straight line by placing one foot directly in front of the other.

The second case was that of a boy of 18, whom I saw through the kindness of Mr. Kelloch. His chief trouble was a sore on the left ankle and deformity of both feet. The sore he had had as long as he could remember, and it was evidently of a trophic nature, but it had only recently become painful. There was well-marked pes cavus of both feet, and all the toes were in the hammer position.

The knee-jerks were increased, and both plantar reflexes gave the extensor response, but the left more so than the right. There was a very slight lateral nystagmus on fixing the eyes, but no definite ataxy.

No other members of the family were affected.

The third case was admitted into Broderip ward under the care of Mr. Henry Morris, who very kindly asked me to see him.

The patient was a man of 21 years, a blacksmith by trade. He was an only boy, but had six sisters, all of whom were well.

He was admitted on account of two small ulcers on each foot which would not heal. These he had had for six years, *i.e.*, since the age of 15. The first to appear was a small ulcer on the right foot, and this was soon followed by one on the left. The right foot had recently altered in shape, and he said the ground felt soft, and that he could not walk in the dark or stand steady with his eyes shut. He had not had any trouble with his sphincters.

On examination there were two small but rather deep ulcers on the inner side of the sole of each foot as above mentioned. The knee jerks, elbow and wrist reflexes were all increased, and there was a well marked jaw jerk. Both the plantar reflexes were extensor in type.

The pupils were normal, and there was a very slight lateral nystagmus. There was slight incoördination of the legs, but no anæsthesia of any kind. There was no tremor and no curvature of the spine.

The right foot was in a position of equinovarus, and there was very marked pes cavus of the left, so that walking was impossible. In order to try to improve the position of the feet, Mr. Morris divided the tendon-Achilles of both feet, but as a sufficient improvement in the position was not obtained by this means, it was decided to do something further, and so a wedge-shaped

piece was taken out of the tarsal bones of each foot, and when this was done the bones were found to be light, porous and brittle, just as they are in the osteopathies of locomotor ataxy.

The fourth case was that of a boy aged 10 who came to the hospital complaining of difficulty in walking, and on examination was found have marked pes cavus of the left foot, which had been gradually coming on for seven years, *i.e.*, since the age of 3. Both knee jerks were exaggerated. The plantar reflex gave an extensor response on the left side and a flexor one on the right. There was a little difficulty in standing with the eyes closed, which was stated to have been noticed for the last two years. There was no loss of sensation, no nystagmus, no spinal curvature, and no change to be seen in the discs.

He had two brothers, aged 8 years and 15 months, and one sister, aged 3 years, all of whom were said to be healthy.

The fifth case was that of a man aged 42. He showed great deformity of the feet, which had been gradually progressing for more than twelve years. The knee jerks were both greatly exaggerated. There were no other definite symptoms. No other members of the family were affected.

Before these can be accepted as isolated cases of the family groups already referred to, it must be shown that they do not correspond to any other recognised groups.

Comparing them with Friedreich's cases it will be seen that while in some respects they closely resemble it, they all differ at once in the matter of reflexes, and also in some cases in the presence of trophic changes in the skin and bones.

It might, perhaps, be expected that here and there a case of Friedreich's disease would be found to have increased reflexes, but the loss of these has always been looked upon as such an important matter in making a diagnosis, that it is not possible to look upon all these as accidental exceptions. All writers speak of an increase of deep reflexes as most exceptional. Osler quotes Strümpell to the effect that this loss is, next to ataxy, the most important sign of the disease.

Sir William Gowers, in speaking of the knee jerks in Friedreich's disease, says, "very rarely it has been increased, chiefly in untypical cases."

In the article in Clifford Allbutt's "System" it is stated that "the so-called deep reflexes are invariably absent from the outset."

Ladame says, "the abolition of patellar reflexes is an absolute rule in this disease, and one will do right always to mistrust the diagnosis of this disease whenever the knee reflexes will not be lost, although all the other symptoms would seem to confirm it."

In the face of these expressions it is clear that these cases differ from those of Friedreich and cannot legitimately be classed with them.

Turning to Marie's cerebellar cases it will be seen at once that there is a very important difference in the presence of the club foot, which is such a constant and early symptom in my cases, but which, as will be seen from the passage I have quoted earlier in the paper, is not recognised by Marie as occurring in his cases at all. There is also evidently a tendency for the disease to begin at an earlier age than in Marie's cases.

Syringomyelia has been excluded by the absence of any characteristic or suggestive sensory changes, and the age, with the exception of the one elderly man, and mode of onset, taken with the other symptoms, enable a differentiation to be made from disseminated sclerosis.

It might, perhaps, be suggested that the elderly man was a case of ordinary primary lateral sclerosis in an adult, and not bearing any special relation to the family group, either as an isolated case or otherwise, but the onset and course were so characteristic that I have not hesitated to record him with the others.

I therefore suggest that these cases, which at first sight are like Friedreich's disease with exaggerated reflexes, are the isolated instances of the family condition known as hereditary spastic paraplegia, and that they are in every way comparable to the isolated cases of Friedreich's disease, to which they are closely allied, but from which they differ chiefly in the predominance of the lateral over the posterior sclerosis.

There is one point which deserves notice in comparing

my cases with the family ones that have been recorded, and that is the presence of a slight amount of ataxia in some which appears to have been absent from the family ones. This presence of some degree of ataxia in certain cases can hardly be regarded with surprise, for although there is at present no *post-mortem* evidence to offer on the subject, the clinical picture is such as would lead us to expect that the posterior columns would also be sometimes involved. Indeed, it is probable that considering the relation which this disease bears to Friedreich's disease there are probably the same changes underlying both, but that in the one the posterior sclerosis, and in the other the lateral sclerosis, forms the predominating feature of the case.

So close, indeed, is this relationship that the two types may sometimes actually appear in the same family.

Ormerod[1] has recorded such in his paper on "Some Further Observations on Friedreich's Disease." The patient was the mother of two children both of whom suffered from Friedreich's disease. The mother had a form of spinal disease which she herself thoroughly identified with that of her children, but as there was absence of nystagmus, speech affection, and ataxia, Ormerod hesitated to strictly class it with the others.

Development had been going on for thirty years, although it was thought that the symptoms might possibly have been modified latterly by a fall. The ribs projected in the left mammary region suggesting spinal curvature, but through her wraps none could definitely be made out.

Knee jerks and ankle clonus could be obtained on both sides. There was rigidity of the lower limbs.

Lateral sclerosis was evidently the main feature of this case whatever group it is put into, and Ormerod in speaking of it says: "It need not, perhaps, surprise us to find symptoms of lateral sclerosis in the mother, as opposed to the symptoms of posterior sclerosis in the children, if we remember, that such knowledge as we possess of the morbid anatomy of the disease goes to show, that both lateral and posterior columns are affected in the course of time, albeit

[1] BRAIN, 1888, vol. x.

the posterior are affected primarily and most completely." More striking still is an instance recorded by Dr. Howard Gladstone,[1] where two brothers both had typical symptoms of Friedreich's disease, but while in one case the deep reflexes were lost, in the other the knee-jerks were exaggerated and ankle clonus was present.

To sum up then we have a disease which clinically shows itself by a premature degeneration of the lateral tracts with the posterior, and possibly other columns occasionally participate to some extent ; it has a tendency to run in families and apparently, though to a less extent, to be actually hereditary ; isolated cases are also frequently met with. There is a very close relationship between it and Friedreich's disease, but it is distinguished from the latter by the predominance of the spastic symptoms, although here and there as might be expected the two practically merge into one another.

[1] Brain, vol. xxii. p. 615.

A REPORT OF TWO CASES OF PARAPLEGIA OCCURRING IN VARIOLA, ONE BEING A CASE OF ANTERIOR POLIOMYELITIS IN AN ADULT.[1]

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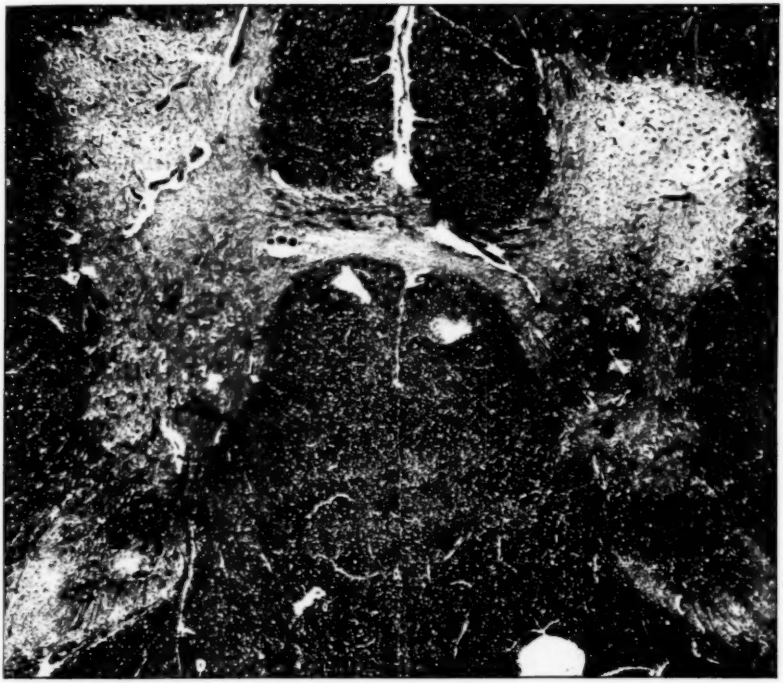
My interest in the subject of paralysis caused by variola has been aroused by the study of the spinal cords sent to me by Dr. Jay F. Schamberg from two cases of variola in which paraplegia of the lower limbs had occurred. It has seemed important to determine whether paralysis is a frequent complication of variola, and what is known concerning its clinical form and its pathology. Dr. Schamberg, who, as visiting physician to the Municipal Hospital, has had an exceptional experience in the study of variola during the present epidemic, informs me that in 2,800 cases of variola observed in the Municipal Hospital during the past two years, there were seven instances of paralysis complicating the disease. In one patient, a woman, the paralysis appeared during the initial stage. She was brought into the hospital in a stuporous state, barely able to articulate. She had great difficulty in swallowing, and impaired power in the arms and legs; the loss of power in these members later became almost complete, but gradually a restoration of function occurred. She had a most pronounced scanning speech which was still present when she was discharged from the hospital. The reflexes were markedly exaggerated.

[1] Read at a meeting of the Section on General Medicine of the College of Physicians of Philadelphia, November, 1903.



Photograph of a section from the lumbar region, stained by the Weigert hæmatoxylin method. The degeneration is almost confined to the anterior horns. These are much less stained than the posterior horns, and the small black lines seen in them are chiefly blood-vessels.

Another patient, a young coloured man, had an extensive gangrene of the external genitalia. At the end of about ten weeks from the onset of the disease he developed partial loss of power in the legs and arms. He could walk with great difficulty with a cane. This condition persisted to the day of his departure.

A third patient, a child one and a half years old, is not included in the cases referred to above. On the day before the appearance of the eruption she developed a hemiplegia of the left side following protracted convulsions.

Of the seven cases referred to, three recovered and four died.

Some of the cases reported in literature presented the clinical appearance of acute ascending paralysis, although they were not always typical examples of this affection, and some were cases of anterior poliomyelitis. We should expect to find myelitis or multiple neuritis as the cause of the paralysis, but in several cases no lesions could be found. Although the occurrence of paraplegia in variola has been known many years, I have not been able to find many reliable cases reported; it is possible, however, that I have overlooked some.

Concerning the implication of the nervous system in variola, H. Immerman,[1] in his monograph on variola, says that the nervous system is relatively often affected, either during or after variola, and is affected in manifold manner. Delirium is common. Purulent meningitis is a rare complication. Encephalitis, cerebral softening, and hæmorrhage are also rare, and usually cause no clinical signs. The spinal cord is more frequently affected than the brain, and motor paraplegia is produced, and this paralysis may occur in the mild or severe forms of variola, and at any stage of the disease; in the incubative or initial stage, or in advanced convalescence. The paraplegia usually develops rapidly, and is generally confined to the lower limbs. The bladder and rectum may be affected, but sensation usually is undisturbed. Sometimes the paralysis is rapidly ascending, like

[1] H. Immerman, "Nothnagel's Specielle Pathologie und Therapie," vol. iv., part iv., p. 89.

that of Landry's paralysis. Death is the common termination. Acute ataxia (Westphal) is more infrequent than paralysis, and monoplegia like that of anterior poliomyelitis is still more infrequent.

As far back as 1857, Raoul Leroy d'Etiolles[1] reported a case in which paraplegia occurred during the incubation of variola. The patient was a woman, aged 21 years. After she had been feeling badly a few days she became weak in the lower limbs. Sensation was not affected. The case was then recognised as one of variola. Death occurred although the palsy had become less severe. No lesions were found in the central nervous system.

Another case that he reported at the same time was as follows: Complete paralysis of motion and sensation developed rapidly in the lower limbs during the period of desquamation, and was accompanied by severe pain on percussion over the spine. The bladder and rectum were paralysed. These symptoms developed when the patient appeared to be recovering rapidly. The left upper limb also became paralysed. No lesions were found in the central nervous system.

Adolphe Gubler,[2] in 1860, reported that in two cases of paralysis in variola in which a *post-mortem* examination was made, no lesions were found in the central nervous system.

In a case reported by Damaschino,[3] a child aged 2½ years had variola. During convalescence the lower limbs became weak. When examined six months after the attack of variola the affected muscles of the lower limbs were flaccid and had lost the electrical contractility. Sensation was intact, and no pain was felt. A focus of softening was found in the anterior part of the gray matter on the left side in the lumbar region, and the nerve-cells of the anterior horns were atrophied. The alteration in the cervical and thoracic regions was much less intense than in the lumbar region. This case probably should be regarded as one of poliomyelitis caused by variola.

[1] Raoul Leroy d'Etiolles, "Des Paralysies des Membres Inférieurs." Paris, mdcccLvii., p. 93, part second.

[2] Gubler, *Archives Générales de Médecine*, 1860, vol. i., p. 537.

[3] Damaschino, *Gazette Méd. de Paris*, 1871, p. 505.

In Bernhardt's[1] case, a man, aged 29 years, during convalescence from variola, became gradually weak in the lower limbs, and all the extremities soon became very paretic and flaccid. A microscopical examination of the spinal cord and nerves revealed nothing abnormal.

In the case observed by J. U. Chalvet,[2] a man, aged 27 years, had variola. The second day after the development of the eruption the lower limbs became weak, and within a short time all the limbs became paralysed. The muscles were flaccid. Sensation was affected in all four limbs. Pressure over the spine in the lumbar region was painful. Death occurred a few days after the development of the paralysis. The spinal cord was found to be very vascular, and nerve-cells of the anterior horns were swollen. This case was regarded as one of acute ascending paralysis.

C. Westphal,[3] in 1872, reported several cases of variola or typhoid fever in which symptoms—scanning speech, ataxia, tremor, &c.—resembling those of multiple sclerosis occurred. The cases were without necropsy, so that the character of the lesions could not be determined. The following year A. Otto[4] described a case in which ataxia, tremor, disturbance of speech, mental symptoms, &c., were observed following variola. The patient was not paralysed. This case also was without necropsy.

In one of the cases of paralysis from variola, reported by C. Westphal, in 1874 (Case 1), the gray matter of the cord was most affected, and was infiltrated by fatty granular cells. In some of the sections the gray matter alone was diseased. Where the gray matter was intensely affected the white matter did not escape. This case seems to resemble closely the second one that I report, and may be regarded as one of poliomyelitis.

In another case diffuse myelitis was found.

Westphal called attention to the mild form of variola which was followed by paralysis, so mild in his first case

[1] Bernhardt, *Berliner Klin. Wochenschrift*, 1871, p. 561.
[2] Chalvet. *Gazette des Hôpitaux*, Aug. 5, 1871, p. 369.
[3] C. Westphal, *Arch. für Psychiatrie*, vol. iii., 1872, p. 376, and *Idem*, vol. iv., 1874, p. 338.
[4] Otto, *Allgemeine Zeitschrift für Psychiatrie*, 1873, vol. xxix., p. 335.

that the patient was not obliged to go to bed during the early stages of the disease.

The report of a case by Gros, in 1883, I have been unable to obtain.

Paralysis occurring in variola has been observed by Oettinger and Marinesco.[1] The disease at first appeared to be of a very mild type. The patient, a man aged 20 years, who had been vaccinated in childhood, and still had the scars of the vaccination, was taken sick on December 11, 1893, and on December 14 a slight eruption appeared. On December 19 the temperature rose, and urination became impossible. The following day the lower limbs became paralysed and flaccid; the upper limbs were not affected. The patellar reflexes were lost. Tactile, thermal, and pain sensations were much impaired in the lower limbs and in the abdominal walls. On December 21 both upper and lower limbs were paralysed and flaccid, respiration was irregular, and the patient was unconscious. He died December 23. The vessels of the pia were injected, and the spinal cord in the lumbo-thoracic region was so soft that it was removed with difficulty. Nerves of the lower limbs examined microscopically appeared to be normal. Both gray and white matter of the lower thoracic region were affected, but the gray more than the white. Perivascular infiltration of mononuclear and polynuclear leucocytes was pronounced in this portion of the spinal cord, and some of the infiltrating cells and even nerve-cells contained micrococci. Small hæmorrhages also were present in the nervous tissues. Perivascular cellular infiltration was also found in the brain.

The clinical histories of the two cases observed by Dr. Schamberg as sent to me are as follows:—

Case 1.—Mrs. H., married, aged 19 years, was admitted to the hospital with smallpox of considerable severity. She was progressing well when during the third week of the disease she became unable to move her legs in bed. Sensation was impaired, but not entirely lost. She had loss of control of the bladder and rectum. She had either no pain at all or very slight

[1] Oettinger and Marinesco, *Semaine Médicale*, 1895, p. 45.

pain. Within a few days partial motion was restored in the lower limbs. At the end of about eight or nine weeks diarrhœa set in, and the patient died.

Case 2.—J. W., aged 38 years, was admitted to the Municipal Hospital on January 13, 1903. He exhibited one fair and two poor vaccine scars from vaccination in childhood. The smallpox eruption was scanty, and distributed in the following manner: About a dozen lesions were on the anterior surface of the body, a few were scattered sparsely over the extremities, and on the face there were about fifty lesions. The patient entered the hospital on the fifth day of the eruption. The temperature was 99° F. Within the next few days the temperature fluctuated from 99° to 102° F. for a week, then dropped to normal.

On January 16, or three days after admission, loss of power was observed in the lower extremities, so that the patient was unable to raise the limbs from the bed. Sensation was impaired but not lost; there was no pain. The bladder was distended to within one inch of the umbilicus. Bowels were constipated. The mental condition was good. About this time the patient vomited.

January 18.—The patient is able to-day to draw up the legs slightly.

January 19.—Motion is better in the legs, and sensation is restored; the patellar reflex is absent.

January 20.—The patient is able to-day to raise the right leg from the bed, but cannot raise the left leg. Catheterisation has been necessary for the past four or five days.

January 28.—The patient passed urine last night for the first time, also had spontaneous bowel movement (before this time the bowels were moved only by enemata).

February 3.—He has incontinence of the urine and fæces, and has much diarrhœa.

February 8.—He has had during the past ten days high irregular fever, fluctuating between normal and 104° or 105° F. There is emaciation, and it is evident that the patient is losing ground.

February 13.—The patient died to-day, this being the thirty-sixth day of the disease.

He had not manifested pronounced loss of power in the upper limbs during his illness.

The necropsy showed the kidneys to be about twice the normal size, and on incision they were found to contain a number of abscesses about one centimetre in diameter. The pelvis of the

kidneys was the seat of a purulent inflammation. The other organs were apparently normal. The cord was removed for microscopical study, and cultures were made from the intradural fluid. An examination of these by Dr. Gildersleeve revealed the staphylococcus pyogenes albus.

Nothing distinctly abnormal could be detected in the spinal cord of the first case. The material had been hardened in alcohol, and the brain and peripheral nerves had not been preserved, so that the microscopical study was necessarily incomplete.

The spinal cord from the second case was very interesting. The gray matter was chiefly affected, and in the lumbar portion of the spinal cord the white matter was scarcely implicated at all. In the lower cervical region a small area in one anterior horn of one of the sections could be found staining a very light colour by Weigert's hæmatoxylin method, showing that the medullary sheaths of the nerve-fibres within this area had disappeared. The white matter was only slightly implicated. Some black spots were found by the Marchi method scattered over the transverse sections, indicating diffuse degeneration from lesions lower in the cord.

About the mid-thoracic region the gray matter of the anterior horns was chiefly affected, and portions of it stained very faintly by the Weigert hæmatoxylin method, but in this region the white matter was considerably implicated.

In the lumbar region the light colour of the anterior horns by the Weigert hæmatoxylin method was in striking contrast to the deep staining of the posterior horns. The white matter was only slightly affected.

Masses of fatty granular cells were found in the faintly staining areas, and perivascular cellular infiltration also was present. The blood-vessels of the anterior horns were congested. The anterior and posterior spinal roots stained well. The nerve-cells of the anterior horns in the cervical and lumbar regions presented more or less chromatolysis, displacement of the nuclei, and loss of dendritic processes, but the alteration of the nerve-cells was not intense.

The degenerative changes of the anterior horns through-

out the cord, causing faintly staining areas by the Weigert hæmatoxylin method were remarkable, and justify the classification of this second case under the head of anterior poliomyelitis. The contrast between the light anterior horns and the dark posterior horns in stained sections was so intense that it was perceptible to the naked eye in the lumbar region. Strictly speaking the case was one of diffuse myelitis, but, with the exception of a part of the thoracic cord, the myelitis was almost confined to the anterior horns, and was an anterior poliomyelitis, and probably of vascular origin.

It seems hardly necessary to insist on the occurrence of anterior poliomyelitis in the adult, but if anyone is sceptical regarding such occurrence, I refer him to the excellent paper on anterior poliomyelitis in the adult by E. W. Taylor.[1] I have already, in association with De Witt H. Sherman,[2] reported a case of polioencephalomyelitis in an adult, but in that case the white matter was more affected than in the second case I report here, and the perivascular cellular infiltration was much more intense. I have discussed anterior poliomyelitis in the adult in that paper.

Cases of anterior poliomyelitis in the adult with necropsy are very rare, as van Gehuchten[3] has recently remarked in reporting a case of this kind. The cases of Damaschino and Westphal (Case 1) and the second one I describe here show that variola may be one of the causes of anterior poliomyelitis.

[1] E. W. Taylor, *Journal of Nervous and Mental Disease*, August, 1902, p. 449.
[2] Sherman and Spiller, *Philadelphia Med. Journ.*, March 31, 1900.
[3] Van Gehuchten, *Revue Neurologique*, August 31, 1903, p. 848.

THE EVOLUTION OF CONSCIOUSNESS.

Together with a Diagram illustrating Certain Homologies in the Nervous System.

BY W. H. B. STODDART, M.D., M.R.C.P.

CONSCIOUSNESS is the faculty or function by which we become aware of the existence of our surroundings. It is obvious that we can only become aware of our surroundings by means of sensations aroused by them. A man in whom no sensations can be aroused is unconscious, he has no consciousness; and *vice versa*, a conscious individual is constantly experiencing sensations. During deep sleep no sensations are perceived; consciousness is in abeyance. The dependence of consciousness upon sensation is illustrated by Strumpell's remarkable patient, whose only communication with the outside world was by means of one eye, the other eye being blind, both ears deaf, and the whole surface of the body anæsthetic. Whenever the sound eye was closed, he went to sleep; in other words, he lost consciousness. Without sensation, therefore, consciousness cannot exist; the essential basis of consciousness is sensation.

The sensations experienced by any given individual can never be experienced by another individual. *I* know that *I* feel and see and hear, but it is pure inference on my part that anyone else can feel and see and hear. The inference is admitted to be justifiable, because other people respond to stimuli in much the same way as I do. Similarly we believe that the lower animals experience sensations. This will readily be admitted of vertebrates; but if it be suggested that invertebrates experience sensation, we are at once told that fitness of response to a stimulus does not entitle us to infer that the animal has experienced a sensation. But

there seems to be an inconsistency in this assertion, for fitness of response is as much evidence of sensation in one animal as it is in another; therefore, it is as much evidence in an amœba as in a man, and he who denies sensation in an amœba should, to be consistent, deny sensation in his fellow men.

The suggestion that an amœba experiences sensation is nothing new. There are many observations, especially by Carter and Romanes, which suggest that an amœba not only has sensation, but that it even possesses such highly developed mental attributes as reasoning and memory. But all that I now wish to claim for the amœba is that it feels, has sensation, and that it knows that it feels, is conscious. A thing which feels and does not know that it feels is inconceivable.

But sensation and consciousness are not the only functions of this unicellular organism. The cell which constitutes an amœba has to carry out the functions of digestion, excretion, movement, reproduction, &c. In multicellular organisms, such as man, cells or groups of cells (organs) have become specially modified to perform these respective functions, and there is division of labour. Some cells (muscle) carry out the movements of the organism, and this is practically their only function; some cells (renal, &c.) discharge effete products from the organism, and this is practically their only function; some cells (ovary and testis) subserve the function of reproduction and some (neurons) the function of sensation, and in each case it is practically their only function. In each case all other properties of the animal cell have relatively diminished to a mere residuum in favour of the properties necessary to the special function of the cell.

On the other hand, the fact must not be overlooked that whilst these residual properties of the cell are considerably reduced, there is no reason to suppose that they are completely obliterated. All cells form definite chemical substances within themselves and discharge them. The muscle-cell is by no means the only contractile cell of the organism; it is only the *most* contractile cell. And I submit

that the neuron is not the only sensitive cell of the organism, it is only the *most* sensitive cell. All the cells of the organism are sensitive to some extent; but sensation is, so to speak, specially worked up in the neuron. Again, if we have any right to say that an amœba has sensation, we are also justified in supposing that each individual of that vast colony of "amœbæ," which we call a man, also has sensation.

Sensation in an amœba has, presumably, only a rough resemblance to sensation in a man, and the same must be said of each individual cell of the human organism. There are great differences even among human beings in the acuteness of their sensations. For example, a highly-educated man experiences extreme pain during the extraction of a tooth, while a navvy scarcely winces at the operation. Lower animals are still less sensitive: Chauveau and Kauffmann were able to operate on the parotid region of a horse while the animal unconcernedly continued a feed of oats, and the presumption is that sensation is extremely elementary in an animal so low in the scale as an amœba, and, with the exception of the neurons, still more elementary in the cells of the human organism.

It has been noted above that consciousness connotes sensation. I submit the converse to be true, that sensation connotes consciousness. We cannot conceive a thing which feels and does not know that it feels, apart from the hypothetical circumstance of it having never experienced more than one sensation. The conclusion is that every cell of the human organism feels and knows that it feels, that it is a conscious unit, that it has a very elementary mind.[1]

[1] The so-called chemiotactic phenomena observed in the case of leucocytes, which make their way towards an area charged with inflammatory products, suggest that those cells experience the feelings of pleasure and pain to some extent. It has been pointed out by Dr. Mercier that pleasure and pain, which induce the phenomena of attraction and repulsion, are the mental accompaniments of sensations which are respectively beneficial and harmful to the individual or its genus, and that evolution would naturally lead to the development of such tones of feeling. It may therefore be inferred that the leucocyte demonstrates a pleasurable tone of feeling, when it makes its way to an inflamed area. It may be going to its destruction, but it is beneficial to the whole organism that it should do so; just as it is beneficial to the genus "spider" that the male spider should engage in coitus, although it goes to its destruction in doing so.

The sensations received by and through these conscious units are the elements which go to make up human consciousness; and the present communication is an attempt to show how this occurs. In order to avoid misunderstanding, let me at once state that I do not suggest that any part of the physical basis of human consciousness is situated, for example, in the skin; but that the sensations experienced by the skin have an indirect representation in consciousness.

The general plan of the nervous system, so far as the motor arrangements are concerned, was demonstrated many years ago by Dr. Hughlings Jackson. He pointed out that the muscles of the body were first represented in the cells of the anterior horns of the spinal cord and in the nuclei of the motor cranial nerves; that the muscles were next represented (re-represented) in the Rolandic region of the cortex cerebri and that there was a further representation in the physical basis of consciousness, this being situated, in all probability, in the prefrontal lobes.[1] At that time, the mode of arrangement of those parts of the nervous system, subserving the function of sensation, was but little understood; but now that we have data to work upon it will be advantageous to study the hierarchy of sensation in the same way.

In the first place, it is to be observed that there are some cells whose sensation is not represented in the central nervous system at all, *e.g.*, leucocytes and most of the cells of connective tissues. But the sensations of cells of the more highly-organised tissues (skin, retina, &c.) are represented in the following way.

Sensations of epithelial (skin) and muscle cells are first represented in the ganglia on the posterior roots or in their homologue — the Gasserian ganglion—the representation being in some cases furthered by special modification of the nerve ends into tactile corpuscles or muscle spindles. Sensations in the organ of Corti are first represented in the ganglion spirale. For the present I omit the consideration of smell and vision.

[1] In an epitome of this nature, it is impossible to do justice to Dr. Hughlings Jackson's work; but I feel confident that the Croonian Lectures for 1884 are too well known to the readers of this Journal to allow of misconception arising from any crudities of mine.

The sensations occurring in all these ganglia are represented in the gray matter in the floor of the fourth ventricle; that is to say, that the peripheral sensations are re-represented there. The posterior-root-ganglion sensations are represented in the nuclei gracilis and cuneatus, Gasserian-ganglion sensations in the sensory nucleus of the fifth cranial nerve, and ganglion-spirale sensations in the auditory nucleus.

The sensations of these various nuclei are in turn represented (those of the ganglia re-represented and those of the peripheral cells re-re-represented) in the cells of the gray matter at the base of the cerebrum, the cutaneous representation being in the optic thalamus and the cochlear representation in the posterior corpora quadrigemina.

Sensations of the portions of these basal masses of gray matter under consideration are in turn represented (the peripheral sensations re-re-re-represented) in the cerebral cortex, the representation of cutaneous sensations being, I submit, in the parietal lobe, and that of cochlear sensations in the temporo-sphenoidal lobes.

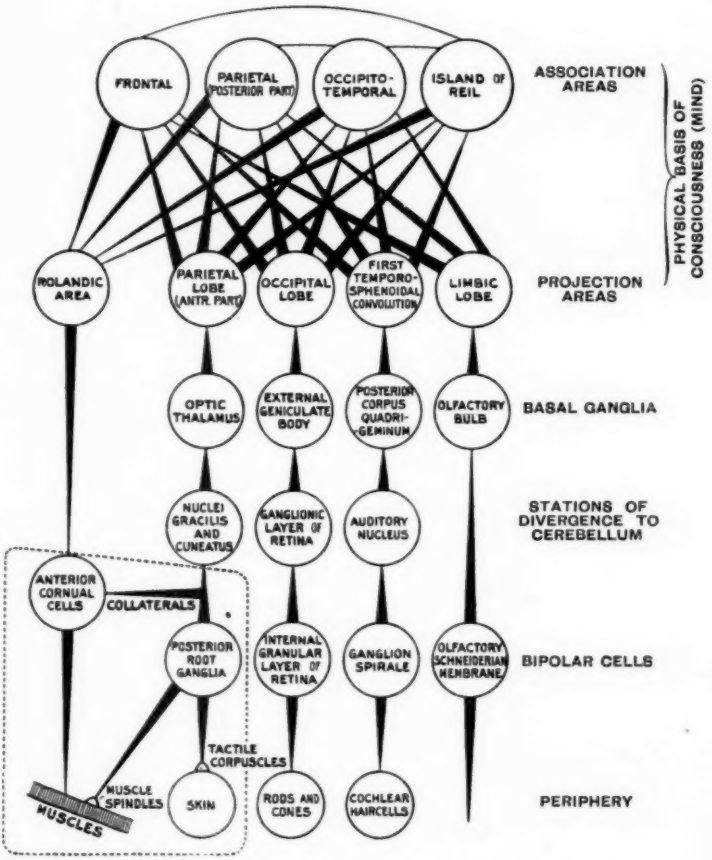
Lastly, the sensations aroused in these projection areas (to adopt Flechsig's terminology) are further represented in association areas (frontal lobes, islands of Reil, the parietal and occipito-temporal areas).

It has been convenient to leave the senses of vision and smell for separate consideration. Sensations of the peripheral visual elements (rods and cones) have their first representation in the bipolar cells of the internal granular layer of the retina, just as the sensations of epithelium are first represented in the bipolar cells of the posterior-root ganglia.

Sensations of the retinal bipolar cells are represented (rods' and cones' sensations re-represented) in the ganglionic layer of the retina. The cells of the ganglionic layer, therefore, correspond to the medullary nuclei of other sense departments.[1] Sensations of the ganglionic layer are in turn represented in the external geniculate body, just as

[1] It will be remembered in this connection that the retina is an outgrowth and hence a part of the central nervous system.

sensations of the medullary nuclei are represented in other gray matter at the base of the cerebrum. Sensations of the external geniculate body are represented (the rods and cones



sensations re-re-re-represented) in the occipital cortex. The highest representation of these sensations is in association areas, especially in the angular gyrus.

For olfactory sensations the cell stations are fewer, perhaps because no connection with the cerebellum exists in the case of smell. Sensations of cells of the regio olfactoria of the Schneiderian membrane are first represented in the layer of mitral cells in the olfactory lobe. Re-representation of mitral cell sensations takes place in the cortex of the limbic lobe and re-re-representation in the association areas.

The appearance of the bipolar cells of the Schneiderian membrane suggests that they are nerve-cells, whose processes extend to the mitral cells of the olfactory lobe. If this be so, there are free nerve endings exposed to the air in the region of the Schneiderian membrane.

The above considerations deal with the senses of touch, muscle-sense, hearing, and smell; the many other senses have been omitted, mainly because our knowledge of their anatomical basis is incomplete.

The accompanying diagram will help to elucidate the above points. The connections figured between the projection and association areas are strictly in accordance with Flechsig's researches, and it will be seen that the cortex cerebri is a colony of neurons, which have very numerous intercommunications. It is a colony of the most sensitive, and therefore of the most conscious, cells of the organism. The combined consciousness of these cells constitutes the consciousness of the colony, and this is none other than the consciousness of the organism.

The projection areas must not be excluded from the physical basis of consciousness until it has been demonstrated that destruction of the association areas results in complete loss of sensation to the organism as a whole. Until this has been proved we must conclude that the physical basis of consciousness consists of the whole cerebral cortex.

Addendum.

It has been observed that fitness of reaction to a stimulus may be accepted as a criterion of consciousness. But fitness of reaction frequently occurs independently of the cerebral

cortex. Such is the case when an ordinary reflex action occurs; this is seen at its best in the classical example of the brainless frog.

When the brainless frog draws its foot away from a stimulus there is a fitness of reaction to the stimulus. And similarly with the other reflexes; there is a fitness of reaction to the stimulus when the eyelid brushes away a particle from the conjunctiva, when sneezing results from irritation of the nostrils, vomiting from irritation of the fauces, coughing from irritation of the larynx, &c., &c. In each case there is an attempt on the part of the organism to get rid of the offending body; yet all these suitable reactions occur independently of the cerebral cortex.

We must therefore conclude that there are many centres of consciousness (colonies of conscious units) in the nervous system other than the cerebral cortex, each of these colonies having a combined total consciousness of its own. One such colony is marked off by the dotted line in the diagram; it is the colony subserving the function of the skin reflexes.

These colonies are severally the physical bases of the consciousness of separate individual parts of the organism, and are no part of the physical basis of consciousness of the organism as a whole. The consciousness of a man is the consciousness of no other part of his nervous system than his cerebral cortex, although there are many centres of local consciousness scattered throughout the organism.

LESION OF THE BRACHIUM PONTIS WITH DIVISION OF THE TRIGEMINAL AND FACIAL NERVES.

BY WM. CHARLES WHITE, M.D.

Professor of Pathology and Neuro-Anatomy, Central College, P. and S., Indianapolis, Ind.

R. E. M., aged 21, enlisted as a private in C Company, 2nd Regiment, U. S. Infantry, in March, 1899, at the age of 18. On March 13, while lying in his bed, he was the subject of the following accident. A fellow private was working with a U. S. A. magazine rifle, in which happened to be a 32-calibre revolver cartridge; during the manipulation the instrument was discharged, the bullet from the cartridge striking M., in his prone position, about the centre of the chin, pierced the skin, inferior maxilla and tongue, and lodged, as found later, at the right side of the foramen magnum.

The notes made at the time of the accident are not available at the present time, and all that is here stated has been obtained from the patient and his father. Only those items which seem to be relevant to the case will be recounted.

After the effects of the shock had worn away, *i.e.*, in four or five days, the symptoms were: paralysis of the right side of the body, face, arm and leg, loss of the faculty of speech, slight loss of hearing on the right side. His right eye was turned upwards and to the left, but he still retained his vision on this side. Swallowing was performed with difficulty, and the sense of taste was disturbed from the start.

His general condition improved fairly well, and in one month's time from the accident he had recovered from the

paralysis of the right arm and leg. How complete this paralysis was could not be determined, and possibly the better way would be to dismiss it here as consequent upon effusion or hæmorrhage pressing on the crossed tract on the right side after the decussation, which effusion was afterwards absorbed. The most likely place for hæmorrhage to collect and produce pressure from such a wound as we are dealing with would be between the clivus of the pars basillaris of the occipital bone and the cord immediately over the pyramidal tracts, after their crossing.

In August, 1899, the following notes were made at the Columbus Barracks Hospital and sent to me through the kindness of Major and Surgeon G. L. Edie, U.S.A. :—

"Gunshot wound of the face, paralysis of the right eye and side of the face, total loss of vision in the right eye and degeneration of the globe; accident, wound received at Annison, Alabama, March 13, 1899. The missile was a 38-calibre, entered the chin, passed upwards into the skull where it lodged at the right side of the foramen magnum, as shown by the X-ray picture; the latter being now on file in Washington."

To the right of the foramen magnum is not a very definite location for the bullet, but is comfirmatory in the light of what here follows.

While yet in bed he constantly rolled toward the left side, even at times rolling out of bed by turning in this way. When he was able to get out of bed in the latter part of April, 1899, he always did so by turning to the left side, and when up constantly staggered toward the left in attempting to walk; this tendency has gradually improved up to the present time, but it still continues in a slight degree, and he always desires to roll out of bed on the left side.

In October, 1899, owing to the inflammation and its results in his right eye, which had not been protected from dust and irritating particles, this was removed by operative procedure and a glass eye substituted.

The writer first saw the patient in June, 1902; at this time he presented the following condition: a little tendency in walking to move to the left side; the trunk, arms, and

legs present nothing abnormal; the right side of the face was quite immobile, and on this side the skin had a flat, thin appearance, extending just to the middle line; the right corner of the mouth drooped considerably, and saliva dribbled from it on to the chin without the patient's consciousness; the right lower eyelid drooped, and tears trickled down on the cheek without producing any sensation; he spoke with some thickness of expression; he did not close his right eye completely, and the right ala nasi moved slightly on deep respiration; the lower jaw when opened projected slightly to the right side; the head movements were normal; swallowing was performed without difficulty or apparent disturbance; the tongue was protruded slightly to the left, but its movements otherwise were not disturbed. All voluntary movements of the muscles of the right half of the face were impossible; there was complete anæsthesia of the skin for sensations of touch, pain, heat, and cold, over the right side of the face, extending from a line corresponding to a perpendicular to the angle of the jaw behind to the middle line in front, and from the anterior-posterior middle line of the head above to the lower margin of the ramus horizontalis maxillaris below; the ear was still sensitive. There was also anæsthesia of the right conjunctiva, the mucous membrane of the right nasal cavity, the right half of the buccal cavity, including the mucous membrane covering the gums above and below, the right anterior two-thirds of the tongue, the right half of the soft palate, and the right posterior wall of the pharynx. The posterior one-third of the tongue was sensitive. The mucous membrane over these parts was smooth and devoid of the small lines found on the other side; this was especially noticeable on the lips. The sense of taste was gone on the anterior two-thirds of the tongue. The sense of smell was at times impaired on the right side, but at other times was all right, depending, probably, on the dryness of the mucous membrane.

For the sense of hearing, air conduction on the right side was almost completely gone and had steadily decreased from the time of the accident to the time of the examination; bone conduction on the right side, however, remained nearly

as good as on the opposite side. Examination of the internal ears presented only some retraction of the drum on the right side.

The loss of the right eye, which has already been referred to, was not due to the loss of sight primarily, but to inflammatory processes secondary to the loss of sensation in the conjunctiva. The left eye was quite normal in vision and movements.

To sum up then we have (1) a complete lesion of the trigeminal nerve on the right side, from its very completeness evidently between the semilunar ganglion and the entrance of the root into the pons varolii, *i.e.*, during its course inwards along the middle cerebellar peduncle; (2) a complete lesion of the facial nerve, distal to its cells of origin in the medulla—a lower motor neurone lesion, from its completeness and the subsequent atrophy of the muscles; (3) possibly a lesion of the eighth nerve. Dr. L. F. Page examined the ear and reported it normal; and yet there was some deafness at first which gradually increased until at present air conduction is entirely destroyed. If any portion of the auditory nerve was injured, it must have been the anterior and mesial or vestibular branch, but as this has nothing to do with the sense of sound, one must look for some deeper lesion in explanation of the absent function of air conduction on the right side.

This would be possible in a lesion of the superior olive, the lateral lemniscus, or the trapezoid body. Only the first of these would conform in position to the course of the bullet, taking its entrance to the pons and its present position as shown by X-ray picture as the points through which to draw a little to indicate its line of travel. The lateral lemniscus would then lie too far cerebralwards, and the trapezoid body too near the median line to be affected. According to Babinsky, if the cochlea be destroyed in a newborn animal there results atrophy and disappearance of the cells and white matter of the nucleus olivaris superior of the same side; and von Bechterew has described a connection between the superior olivary nucleus and the nucleus of the sixth nerve. This latter connection might possibly

explain the muscular derangement of the eyeball in the present case in which the ball was turned upwards and inwards.

Now the only place where such a complete lesion of these two nerves could occur with the present source of injury, *i.e.*, a revolver bullet approximately three-eighths of an inch in diameter, must be at a point where they come nearly enough together to be severed by the bullet in its passage. Only one such place exists, and that is over the brachium pontis, as it sinks into the cerebellar substance. One must remember here that the lesion was practically confined to the fifth and seventh nerves. The fifth nerve as it passes inwards and backwards from the Gasserian ganglion to enter the pons lies closely enough to the seventh nerve, as it passes upwards and outwards to enter the internal meatus, to be divided by such a missile. The possibility of fractured bone causing part of the injury is negatived by the confinement of the lesion remaining.

The course of the bullet entering the chin going through the soft palate and lodging to the right of the foramen magnum admits only of its piercing the tip of the occipital bone to the right of the middle line just internal to the carotid artery as it enters the middle fossa, then severing the fifth and seventh nerves as it passed backward through the brachium pontis on this side, to lodge possibly in the substance of the cerebellum.

One or two features of interest may be noted before discussing the probable lesion of the middle peduncle. The persistence of the sense of taste in the posterior third of the tongue and its loss in the anterior two-thirds on the right side suggest strongly that either one of these two nerves must functionate in the conductions of the sense of taste from the latter region. The late work of Cushing would indicate that the fifth nerve has little to do with this duty, so that the work would be thrown solely on the nervus intermedius which must have suffered division along with the seventh nerve. The lesion of the two nerves being so complete and the function of the posterior third of the tongue being still intact, these nerves have nothing to do in this case at least with the conduction from this region.

The very completeness of the lesion of the two nerves involved by so small a missile is interesting; that they lie so closely together at this point, and that other neighbouring structures of such vital importance should have escaped injury is worthy of emphasis.

The reasons for reporting this case as one of brachium pontis lesion are two-fold, first the similarity which the group of symptoms present in this case bear to those in other reported cases of injury to the same region, and to cases of experimental lesion; second, the impossibility of severing the two nerves, which in this case are evidently divided, at any other point than immediately over the middle peduncle, which must then have suffered injury also.

Ferrier gives as the symptoms of such a lesion, forced movements or actual rotation associated occasionally with deviation of the optic axis. He also recounts the following cases which from their similarity to the present case in symptomatology I feel justified in repeating here. One reported by Longet in which there was softening in the right cerebellar peduncle where it enters the cerebellum, in which there was rotation from right to left. One by Freideburg of hæmorrhage into the left cerebellar hemisphere, followed by meningitis and due to traumatic lesion; the patient suffered from vertigo, a tendency in walking to go to the left, followed by repeated attacks of rotation round the vertical axis from left to right; the eyes were in a constant state of oscillation. One by Bernhardt of a tumour implicating the right middle peduncle in which the patient lying in bed had a tendency to turn from right to left, the eyes looked upward and to the left with some nystagmus, and in walking he had a tendency to fall to the left. One by Nonat of hæmorrhage into the right peduncle, in which the patient assumed a forced position on the right side, with head strongly turned to this side, eyes immovable, right eye looked downward and outward, and the left eye upward and inward. These it will be seen resemble very closely the symptoms in the case of M., reported here, which are referred to the brachium pontis lesion.

In experimental section of the middle peduncle rolling

round the long axis and deviation of the eyeballs are the chief features observed, with, however, a controversy as to the direction of the rotation. The direction of rotation probably depends on the structures injured in the operation, one group of experimenters effecting the lesion from the dorsal, and the other from the ventral surface of the pons.

CEREBRAL EMBOLUS DURING AN EPILEPTIC FIT.

BY WALTER BROADBENT, M.D., M.R.C.P.

THE following case is of interest in connection with Dr. Beevor's experiments on epilepsy related at the end of his Croonian Lectures.

A lady, aged 44, who suffered from rheumatic fever when a child, had been short of breath on exertion for a long time. For seven years she had been subject to epileptic fits; two days before this illness she had five fits. In the evening she was sitting talking to her daughter, when she suddenly was unable to speak plainly and became rigid preparatory to a fit. Her daughter stated that, instead of the usual fit, there was irregular jerking of the right arm and leg and the mouth was drawn over to the right, but she says the head was turned to the left; the left arm and leg remained absolutely passive. She passed water during the fit. She was put to bed and remained unconscious for five hours. When seen she was lying on her back in a drowsy state with slight stertor. She could be roused to answer questions and said she had pain in the right temporal region. Her head was lying straight, but she could not turn it to the left, though she could to the right. Her eyes were both turned to the right, the edges of the corneæ touching the canthi; she could move them to the middle line, but not beyond it to the left The left pupil was dilated, the right normal. The tongue deviated to the left on putting it out. The left naso-labial furrow was flattened, the left arm and leg paralysed, and there was also left hemianæsthesia and almost complete analgesia, a hard pinch being necessary to evoke any response. The movements and sensation of the right side were normal, the knee jerk was absent on the left side, slight on the right. The plantar reflex was present with extension of the great toe on the left side, and normal on the right. The left side of the chest moved poorly in respiration, the right side much better. Temperature a degree higher in the left axilla than in

the right. At the apex of the heart, which was 1½ inches below and ½ an inch outside the nipple, there could be felt a presystolic thrill leading up to a short thrust, and through the stethoscope a rumbling presystolic murmur of variable length, a short loud first sound, a soft systolic murmur and a weak reduplicated second sound could be heard. Her liver extended two fingers' breadth below the costal margin, and there was slight œdema of the legs.

She remained in a drowsy condition and passed everything under her. After the third day the left side of the chest moved distinctly better than the right. About the sixth day some movement returned in the left leg, but no knee jerk could be obtained. She died on the ninth day.

Post mortem.—In the right middle cerebral artery just beyond the point of origin a small embolus was found with a distal thrombus for half an inch. The cortex along the course of the vessel was softened; on section the right corpus striatum and all the surrounding parts, including a portion of the frontal lobe, were in a state of extreme softening.

The embolus must have blocked the middle cerebral artery just at the commencement of the fit, and it illustrates Dr. Beevor's[1] experiment that "no epilepsy was obtained on the same side as the cortex stimulated, when the opposite cortex was removed."

[1] C. E. Beevor, "Croonian Lectures," *British Medical Journal*, July 4, 1903.

Reviews.

Les obsessions et la psychasthénie. Vol. i., pp. xii. and 764, by Prof. Pierre Janet. Vol. ii., pp. xxiv. and 543, by Profs. F. Raymond and Pierre Janet. (Paris: Alcan, 1903.)

In the first volume the preface is followed by analysis of the symptoms. First are taken the obsessive ideas, their content, their common characters, and their form, especially concerning their degree of permanency, of impulsive power, of hallucinatory representation, and the degree of belief and criticism which accompany them. Next, under the general name "forced agitations," are included a vast host of symptoms with their divisions, sub-divisions, re-sub-divisions, and re-re-sub-divisions. Then follows a long discussion of so-called psychasthenic stigmata, also much divided, and ending with a summing up of their general characters Discussion of pathogenic theories follows; then come studies on the lowering of psychological tension, subsequently resumed with those on the hierarchy of psychological phenomena. Next in order are the author's interpretations of the various manifestations of the general obsessive disease, also theories from various sources as to its anatomical representation. Under evolution come etiology and evolution of the disease as to form of onset, principal clinical varieties, course, complications, terminations. Diagnosis, prognosis, with treatment succeed, and the winding up is the place of psychasthenia among the psycho-neuroses. The second volume is mainly occupied by the recital of cases.

To begin with, the work is based upon the study of a large number of persons. This richness of clinical material has enabled the author to speak with greater authority, and to throw into better relief some of the less-studied details, the lesser relative frequency or importance of which have necessarily kept them in the background in published clinical summaries.

The work gives evidence of wide grasp and assiduity in dealing with an intricate, difficult subject, and skilful utilisation of

material to support each of the various views the author has adopted or framed.

Life is short, and we would have liked these huge volumes better had they been of less length. A work so much in the extensive is almost certain to have at least the defects of its excellencies, hence many repetitions, not always self-consistent, and now and again lapses into the diffuse. Moreover, there is much division and subdivision of the subjects, and much seemingly unnecessary naming, and particularly re-naming. Nor are the names always quite happily chosen.

In illustration of methods one may take, for example, the subject of what M. Janet calls "the mental manias" of the disease. The morbid state seeming to him not to be sufficiently explained by the obsessions proper, *i.e.*, the obsessive ideas representing some events, objects, and especially actions, in a general way; he turns to other pathological phenomena which are conjoined with obsession and produce its painful and morbid character. Thus patients may complain that they are forced to think excessively in an exaggerated manner, that their brain works in spite of themselves, that they are forced to make useless movements, to move without any real necessity, or suffer from violent emotions coming on irresistibly, on insufficient provocation. These phenomena may occur without any clearly precise obsessive general idea.

These forced mental operations are not normal; on the contrary are excessive, sterile, and of inferior order. And, adding the word "agitation" as applicable to the whole group, he unites them under the name of "forced agitations," divisible into several sub-groups, by the first of which sub-groups, namely, "mental agitations," he means operations of intellectual order. To the first or "systematic" product of division of this sub-group he applies the name "mental manias." But this name "mental manias" is applied in a popular or unlettered sense, and not, as one would expect to find in a scientific work, in the meaning as employed in psychiatry. For the author does not look upon the subjects of these "manias" as being, in virtue of such manias or of anything else, insane. All through the book he labours to exclude that inference. To those who have been endeavouring for years to bring the word *mania* into more definite, precise, and selective application, it is discouraging to find it applied to so common a use.

The "forced agitations" are divided into three sub-groups; each of these into systematic and diffuse; each of these again

subdivided into a number of kinds; and a further subdivision is made of these into varieties, numerous and bewildering. Mental agitations form the first sub-group, and are divided into systematic and diffuse; of these the "systematic" are the so-called mental manias of the work. From a table and from the text we collect forty "mental manias." And elsewhere are others not mentioned in the section specially devoted to this subject.

Then, surveying at large, one cannot see the forest for the trees; viewing the minutiæ, we muse on a mania of the quest of the atom.

"*Psychasthenia.*"—For the patients affected with the general train of mental besetments and cognate states—by some called "degenerated delirants," or "neurastheniacs," or "phrenastheniacs"—M. Janet formerly proposed the name of "the scrupulous"; thus vastly increasing the scope of a name long ago applied by Ball to one sub-group only; and he now uses the name "psychastheniacs," hoping to bring the diverse symptoms of many diseased states into a single great psycho-neurosis—psychasthenia; indeed, for some time he has used "psychasthenia" to designate the special mental feebleness of the obsessed, in contrast with that observed in hysteria. He calls it "this special enfeeblement of the mind, psychasthenia." He also devotes much space to discussion of psychological and physiological insufficiencies and departures from normal observed in besetments, and labels them "psychasthenic stigmata."

But psychasthenia is a condition far too general and widely inclusive to be thus narrowed down and reduced to the duty of summarising the state of mind fundamental in obsessive conditions. Psychasthenia (asthenia of *psyche*) demands great breadth of application; every general morbid psychosis and neurosis, developing out of the fruitful soil of hereditary morbid weakness or defect, necessarily has its own particular variety of psychasthenia. Obviously there is a number of psychasthenias.

This also brings us to the subject of the relation of besetment to neurasthenia; inasmuch as some have attributed all the phenomena of the former to neurasthenia, and as cerebrasthenia and phrenasthenia are usually placed under neurasthenic headings, and, indeed, as the nervous system and functions—the whole—include the part, the brain and its functions—so neurasthenia includes psychasthenia, and has first right to this as a name pplicable to part of its domain. I straitly limit this

remark to the word as a name, and do not refer to what is described under it in these books; for to do so would be to assume the truth of one opinion on a contested question.

And as to the nosological position and congeners of besetments, a number of different views occur to the mind, such as, (*a*) that besetments are stigmata of mental degeneracy; (*b*) that they are symptoms of abortive or rudimentary paranoia; (*c*) to be taken as sometimes neurasthenic, while different ones occur in degenerates; (*d*) or as practically from neurasthenia, and this last itself a constitutional degeneracy; (*e*) as manifestations of neurasthenia, taken as a great morbid neurosis, which thus is really a psychoneurosis; (*f*) or as an intermediate state between neuropathy and insanity.

The various kinds of mental besetments, or of obsessions in the wider sense of the latter word, have thus been connected with neurasthenia by some observers. And under certain kinds of neurasthenia have been summarily described all the leading mental conditions found in besetments. Naturally these have been especially attached to so-called hereditary varieties of neurasthenia, in which their frequency and their degree are especially great, and in which they are made to constitute an important part of the description of the brain symptoms of neurasthenia, particularly cerebral neurasthenia or cerebrasthenia. In writing of besetments, I have dwelt upon their "close relationships to," and "frequent fundamental association with," neurasthenia.

Are, then, besetments, or the general obsessive disease in its wider clinical sense, to be taken as symptoms of neurasthenia? Or are they merely congeneric; fruits of the same soil, joined with neurasthenia by common neuropathic bonds?

With interest we turn to what the work under review will say, and select relevant passages:—

"What one observes most often are those functional disorders which manifest themselves from infancy, and indicate a defective resistance of the nervous system. . . . . If the physical stigmata of degeneracy are not always evident, there is still a feebleness of nervous system which manifests itself by these functional disorders. . . ." Again, in cases of acute onset "are all the symptoms of neurasthenia more or less pronounced."

Then, at p. 644, neurasthenic states are, for the first time in this book, brought into the classification of the general obsessive disease: namely, as first sub-group of the first group, and consisting of psycho-physiological insufficiencies, with predominance of

organic disorders, constituting the neurasthenic states. Next, under diagnosis, it is observed that when only psychological insufficiencies exist the diagnosis from neurasthenia seems the most difficult to make. Also that, in evolving, neurasthenia can give birth to "psychasthenic states." Then, describing the neurastheniacs most predisposed to that *evolution*, in certain of them the neurasthenic state is spoken of as becoming *complicated* with obsessive conditions. The author next agrees with those who say that the two diseases are two differently localised manifestations of one same nervous exhaustion; and adds that in neurasthenia the exhaustion of the central nervous system is slight, and especially bears on centres of viscera—it augments, extends to the whole brain, becomes more conscious and causes more phenomena of derivation in psychasthenia.

Again, "psychasthenia is a psycho-neurosis very near to neurasthenia . . . . . its place is between epilepsy and hysteria."

Finally (vol. ii.), the first type or group of the persons suffering from "psychasthenia" (in his sense) are those with confused symptoms of physical and mental neurasthenia only; or, as elsewhere, "the simple neurastheniac."

Thus, in the course of the work, there is some doubt and oscillation in the doctrine as to the relationship of besetments with neurasthenia.

The hierarchy of psychological phenomena.—A hierarchy of nervous centres has been a conception long familiar, and involving a corresponding hierarchy of functions. It is a commonplace of the evolutional theory, and in medico-psychological work. M. Janet makes a scheme of the hierarchy of psychological phenomena.

(1) Highest, therein, is "function of the real," exemplified in voluntary action; attentive perception of present external objects with feelings of reality, belief, certainty; and similar perception of one's own real personality. Of these coördinates, the one first mentioned being effective action on reality, social and physical, especially new action, with feelings of unity and liberty; also, being particularly that which enables one to act on external objects and change reality; this is more difficult when it bears on the social medium, or when it is new and requires fresh adaptation to changed circumstance. The second being the higher kind of *attention*—attentive perception with feelings of reality, belief, certainty, as regards the real presence of external objects. The third being much the same, as regards one's own real personality, with feelings of reality and unity.

(2) The next, or second group in the hierarchy of psychological functions, in this scheme, are "disinterested" activities of various kinds.

(3) In the third group in the hierarchy are those concerned in imagery.

(4) In the next are the visceral-emotional reactions; systematic or diffuse.

(5) And the fifth, and lowest, consists of useless muscular movements, systematic or diffuse.

Pyschological tension is brought into relation with the above hierarchy of psychological phenomena; they are hypothesised as fitting higher to higher, lower to lower of each.

Firstly, the unification, the concentration, particularly in a *new* mental synthesis; and, secondly, the number, mass and richness of the psychological phenomena taking part in that synthesis (and, perhaps, their rapidity, or that of their elements), constitute a psychological character which, conventionally, he calls psychological tension.

In a given individual, the degree of psychological tension, or of elevation of the mental level, is indicated by the place in the hierarchy occupied by the highest psychological phenomena of which he is capable.

Says the author: "It is very likely that this psychological tension corresponds to a certain physiological and physical tension in the central nervous system." If by that "correspondence" it is meant that *plus* and *minus* states of all three always go together, in all departments of function, one may incline to differ. We believe in what may be called tensions correspondent (*cet. par.*) with the characters of the psychological activities, and as one element in the attributes of those activities; but these tensions are tension-states and -degrees of the "psycho-physical processes" concerned in mental activities. And it is not merely likely, it seems certain that physical and physiological tensions are necessarily implied and involved therein.

Nor must we deal with the rises and falls of psychological tension as if they were those of a column of mercury rising and falling in a thermometer or barometer. We may take another view of a tension concerned in these, indeed, most complex and highly-evolved processes—processes standing at the summit of human evolutionary attainment on nervous lines.

The writer of this review drew attention to some resemblances between mental besetment and epilepsy, but concluded that they

must be kept separate; and compared and mentioned distinctions between one particular class of cases of each (and not the two diseases, broadly, as seems to have been understood). The subject is one which affords room for very considerable differences of opinion; and it is not in conflict with anything the writer stated when M. Janet says that while he does not identify epilepsy and psychasthenia (in his sense), they have something in common, that their phenomena are neighbours and can easily transform into each other, the common element is the lowering, the fall of psychological tension. But M. Janet eventually goes much further. Playing upon the possibilities of the hypothetical psychological tension and its lowering, he calls the result "a crisis of psycholepsy"; the fall of psychological tension being enormous in true epilepsy, much less in the psychasthenic state, but all the same a fall of consciousness which produces the odd feelings of the patients, their doubts and anguishes. Certain subsequent remarks are not consistent; the same great group is called "the phenomena of derivation *or* crises of psycholepsy"; now for the first time casting into the net of "the crises of psycholepsy" the whole host of the forced agitations and their crises—but a few lines lower, we read, "crises of psycholepsy *with* phenomena of derivation" (italics ours); in a later page the former expression reappears.

Finally, he says: "It seems to me that the psychasthenic state is an attenuated and chronic epilepsy." It is extremely difficult to reconcile this with the statement that he does not identify them, as a moment ago quoted.

And reserve is necessary as to the name "psycholepsy," used in the sense proposed. Psychic epilepsy, or psychical epilepsy, already has an established and well-set place as a modern department of ordinary classical epilepsy; and although the names are not the same, they are so very much alike in appearance and in sound that the adoption of "psycholepsy," in M. Janet's sense, would certainly lead to many misprints, confusions and misapprehensions; especially when transfers, or translations, were being made into other languages.

Not to go back to the history of the medical recognition of the component elements, opinions have differed much, even in fairly recent years, as to the field covered by the relevant morbid states; some excluding cases which one took to have right of admission; others including syndromes which seemed to lack such right. The nomenclature was redundant. The word obsession was by

some restricted to ideas; by others used in a far wider sense inclusive of all related mental products. And it seemed that the place of impulse therein was belittled. Consequently, to the whole group-assemblage of states, which seemed to be rightly placed together, I applied the name *mental besetments* (*Journal Mental Science*, October, 1896), and took them to contain a blending of various defective and abnormal mental states, intellectual, emotional, volitional, but that anyone of these three functional kinds is in some examples the most conspicuous or clinically predominating feature, or forensically the most important; what one had in mind being established, well-developed cases as they come to the physician's examination, and with such histories as they or their friends give.

In a report containing reference to that article the position taken was misunderstood, and, citing therefrom, M. Janet attributes to me adhesion to one of the "intellectual" theories (to these we recur later). But the words he thus cites as showing my view at that time are part of a paragraph containing a brief summary and consideration of the view held by another. This is sufficiently clear from the paragraph itself, and my view was explicitly stated in what immediately followed, namely: "But I think that here the proper organising idea is that, broadly viewed, besetments invariably tend to, and usually are, a blending of anomalies of all three, *thought*, *feeling*, *will*. The tendency is always present; the blending produces the usual and composite state.

"Truly there are cases in which imperative insistent dominating *conceptions* of recurring labile remittent or intermittent fixity hold first place (*i.e.*, in sense of clinical prominence). . . .

"And there are others in which an *impulse to act*, or an irresistible one, seems to be the chief and leading symptom. . . .

"Yet in still other instances *morbid fear*, terror, or horror, is apparently the predominating mental condition . . ."

And in the next two or three pages these views are concisely amplified, and as regards developed cases it is claimed that disorder of one of those kinds alone is practically never the sole pure element of besetment; that an entirely non-emotional variety of obsessive ideas does not exist; that in the emotional states concerned there are intellectual elements; that the obsessive acts of the patient have emotional and intellectual antecedents and accompaniments; and that in many cases of various form there are apt to be the strangest bodily feelings and changes. One statement, however, then made in acceptance of certain experience of Westphal and others, would not have been made later.

A few words as to what M. Janet calls the pathogenic theories of the disease, that is to say, from the point of view of theories dealing with the question as to which symptoms ought to be considered as the most important and as the point of departure of the other symptoms, or as the point of departure only (it is put both ways, p. 446). For from that point of view he divides the theories into intellectual, emotional, and psychasthenic.

One may raise the question of the congruity and validity of division of theories, *on that basis*, into intellectual, emotional, and psychasthenic. The psychasthenic theory being, on that basis, an intellectual and aboulic theory, and swiftly an emotional theory: for, according to the description in the work, the earliest symptoms are partly intellectual, partly aboulic, and at once accompanied with emotional states; the first-mentioned are intellectual, though not consisting of the perfected obsessive ideas or imperative conceptions.

And on another ground reserve seems well. For under the emotional theories, *on that basis*, the book places all theories taking any "forced agitation" as point of departure, namely, all examples in which the comparatively early states and prominent conditions are those of mental manias, mental ruminations, tics, crises of motor agitation, as well as those in which they are the phobias and anguishes.

The relations to insanity.—I mentioned the close relationships of besetments to hypochondria, paranoia, and passage to "fully-developed degenerative psychoses, such as those attended by hypochondriacal or by persecutory delusion," and some cases clinically in part of melancholic type. Also their intimate blendings and transition phases with the phenomena of the insane diathesis simply, with paranoia, imbecility, so-called moral insanity, original paranoia, hebephrenia.

M. Janet makes insanity in the obsessed to be merely a complication by anxious melancholia or by paranoiac states; also mentioning ecstatic states, mental confusion with stupor or excitement, and suicide.

I think this does not go far enough. Besides what is recapitulated just above, I have claimed for some besetments that "in an elaborate and advanced form they pass into a variety of insanity and rule the subject's life"; also that "the transition, psychologically, from the morbid fear or involuntary compulsive feeling or thought, to insanity, especially the insanity of doubt, seemingly consists in a transition from a state in which the patient recognises

(perhaps laughs at) the incorrectness, inaptness, irrelevancy or absurdity of his thought or feeling, to the vesanic state in which he is engrossed by the vivid reality and validity of the feeling or thought, so that by it the life is essentially altered and the acts are largely dominated."

For practical purposes, such a person is insane. Take again my summary of one class of severe cases of besetment, sketched in a few words.

"And hence some of the prolonged wearisome repetitions, doings and undoings, thousand plans and stratagems, attempts and imagined failures, renewed strivings and insistive repetitions; so that torturing hours are consumed in the process of dressing or undressing, of paying attention to the calls of nature, or of doing some other simple everyday act of life—but now done with the wearisome magic ceremonial of morbid precaution, hesitation, and doubting fluttering vacillation."

Or one might take the clinical description of some of the more severe cases in the volumes under review, other than those in which insanity is admitted.

Under the influence of besetments some persons are reduced to expending life very much in aberrant forms of mental and motor activity, or inertia; painful, barren, useless; sometimes directly or indirectly detrimental; not seldom absurd, ridiculous; out of proper adjustment to familial, social and occupational relationships; blighting the life; and in short, constituting a severe derangement of mind and insanity of conduct, from a practical point of view. Indeed, some besetments are worse than are many milder examples of recognised insanity.

There is an argument in the work that obsessive ideas *par opposition à d'autres états délirants*, never present a complete absurdity. But many obsessive ideas which sway the life and dominate the personality of the obsessed sufferer are distinctly absurd, or even very absurd. And, on the other hand, many insane delusions are not absurd, but are false beliefs, due to disease of mind, expressing mere possibilities as achieved actualities.

There are interesting remarks and discussions on the interpretation of the various symptoms of the obsessive disease, for which we have not space. We think that the same general principles of nervous and mental physiology and pathology apply to the interpretation of the morbid states in besetment as hold good for the interpretation of abnormal mental states in the diseases recognised

by all as insanities. In both, there are many degrees, extents, rapidities and kinds of nervous and mental dissolutions; and various relations of these last, *inter se;* with subsidiary effect of attempts at re-integration.

Finally, it may be added that the value of the work is heightened by the fact that M. Pierre Janet has successfully laboured in somewhat cognate psychological fields, and his investigations on hysteria, on neuroses and fixed ideas, and on pyschological automatism, have already greatly enhanced his reputation. And in the second volume we seem to find the expert clinical influence of M. Raymond.

We most heartily congratulate M. Janet, who is author of the greater part, and his colleague in the second volume, M. Raymond, upon the work as a whole, and as an able contribution to the literature and analysis of the large important subject dealt with.

W. Julius Mickle.

Essai sur l'hémiplégie des vieillards (*Les lacunes de désintégration cérébrale*). Par le Dr. Jean Ferrand. (Paris: Librairie médicale et scientifique, Jules Rousset.)

This monograph is an exceedingly interesting one. It deals with a class of cases which, because they are chronic and may not have very obtrusive symptoms, are apt not to receive as much scientific attention as they ought to, and it associates with this clinical condition certain pathological changes of a fairly definite character, differing from those which are the result of hæmorrhage or thrombosis of cerebral vessels. These changes are the formation of cavities or *lacunæ*, and the author has been fortunate enough to observe at the Bicêtre a large number of cases during life and to examine the brains of these patients after death, and to verify the existence of definite pathological changes underlying the clinical phenomena which have been present.

This essay gives the result of his observations—clinical and pathological—in no less than eighty-eight of these cases.

The condition which he found in the brain was the existence

of cavities—*lacunæ*—chiefly in the gray nuclei—the lenticular nucleus, the caudate nucleus, and the internal capsule, in that order of frequency. They also occur in the pons, in the centrum ovale, and the corpus callosum, but very rarely in these last two situations. Dr. Ferrand describes the cavities as of two kinds; (1) the miliary cavity, probably the most frequent, which, on account of its small size and the comparative absence of clinical symptoms, is apt to be overlooked. It is seen with the microscope in a properly stained specimen to consist of a cerebral vessel (with thickened but permeable walls) surrounded by some much thinned tissue. (2) In the larger cavity or cavities, those of the second variety, instead of attenuated tissue around the cerebral vessel, we find a definite loss of substance, the vessel surrounded by a cavity. The vessel is permeable, its walls are thickened, and its muscular coat especially is degenerated and fibrous—the result, it is said, of an arteritis. The walls of the cavity are formed by the cerebral substance itself altered or modified by the disintegration which has taken place, and the cavity contains a quantity of what is best described as cellular and fibrous detritus.

The existence of cavities in the cerebral substance has been long known. Sometimes such cavities are filled with fluid, and are then, of course, cysts—the result usually of cerebral hæmorrhage; sometimes they are really softened areas, the result of vascular occlusion. We also know the cavities which exist in what has been called the Gruyère cheese brain, and these are probably the result of putrefactive changes. The cavities described in this monograph, to which Pierre Marie has the credit of first directing attention, are different from all those, and will, we believe, be found to be characteristic of a large class of cases hitherto not clearly recognised or separated from cases of hæmorrhage and softening. Their clinical characteristics seem to be the history of a stroke usually slight and transient, the persistence of some weakness not necessarily unilateral, a tendency towards improvement, and a long duration of weakness finally terminating in some accidental complication or in cerebral hæmorrhage. The pathological condition corresponding with this is one in which one or more cavities of varying sizes exist, usually in the lenticular or caudate nuclei. These cavities have a vessel in their centre, a vessel with thickened walls and permeable lumen. At first this has granular bodies surrounding it, and the brain substance forming its wall is infiltrated with small cells—is, indeed, in a state indicating some inflammatory process of

a chronic kind. As time goes on the cavity becomes more or less obliterated, but, as can easily be understood, a diseased and degenerated vessel, unsupported as the vessel in such a cavity naturally is, may by rupturing give rise to a large cerebral hæmorrhage and a fatal termination.

The monograph is very clear, comprehensive and convincing. Its value is considerably enhanced by the illustrations, which are mostly photographic, and by the detailed record of the cases—clinically and pathologically—on which it is founded.

JAMES TAYLOR.

Text Book of Anatomy. Edited by D. J. CUNNINGHAM, F.R.S. (Young J. Pentland, 1902.)

THERE is no indication given in the preface of this book as to the purpose for which it was especially designed, but we may take it that the term "text-book" is intended to denote, first of all, a standard work for the use of students in their dissecting and examination periods of life, and secondly, a convenient book of reference which should find a welcome in a library or a laboratory. From either point of view it would be impossible to form a reliable opinion of its merits from a mere perusal of the contents, and it has therefore been necessary to put the volume to its proper test, and to postpone judgment on it until after the lapse of a year, during which time it has been constantly in use as a laboratory work of reference.

The result of this trial is wholly favourable, and endorses the opinion which is the natural one to form of a book of which the list of authors includes such names in the anatomical world as those of Professors Cunningham, Arthur Thomson, Paterson, and others.

We are here chiefly concerned with the parts dealing with the nervous and muscular systems, which have been undertaken by Professors Cunningham and Paterson. About 150 pages have been devoted to the central, and 100 pages to the peripheral parts of the nervous system, and in that comparatively small space an enormous amount of information has been compressed. In spite

of the compression, however, the facts are clearly given, and, with the aid of an admirable index, are rendered easily accessible to anyone looking up a particular question.

Considerable skill has been shown in deciding what should and what should not be omitted, the scope of the work forbidding the introduction of controversial subjects; but perhaps more than two lines might have been devoted to the descending fibres in the posterior columns, and some mention made of the less well-known tracts in the anterior and lateral columns of the spinal cord.

The illustrations are numerous, original and excellent, while the reader is pleasantly surprised to find that in lieu of references to p, p^1, and p^2, the names of the various parts are printed distinctly on the drawings and diagrams.

The chapter on myology is fully up to the high standard of the rest of the work, and the attempt to describe the actions of muscular groups rather than those of individual muscles is one to be thoroughly commended in a work of this kind.

In conclusion, it should be stated that the book is a single volume, containing about 1,300 pages, and that the paper and type are all that can be desired.

E. FARQUHAR BUZZARD.

Les tics et leur traitement. By HENRY MEIGE and E. FEINDEL. (Masson et Cie., Paris, 1902.)

IT is a matter of some surprise to meet with a book of more than 600 pages devoted to the subject of "tics," but it is more than surprising to learn that its authors have, severally or conjointly, already contributed twenty-three other publications to medical literature on the same subject. This is a degree of specialism hardly known in this country, and calculated to raise the question as to whether the particular specialty is of much greater general interest in France than in England. Should the answer be in the positive, it affords strong evidence in favour of the view that this form of malady is of far more frequent occurrence across the Channel.

Those who have the leisure to give to its perusal will find much to interest them in this volume, for not only is the subject

treated exhaustively from every possible standpoint, but it is dealt with in a most attractive manner and in a language which lends itself peculiarly to graphic descriptions of the extraordinary movements characteristic of the disease. One must, for instance, agree with Meige and Feindel that the following summary of the condition by Trousseau is "irreproachable":—"Le tic non douloureux consiste en des contractions instantanées, rapides, généralement limitées à un petit nombre de muscles, habituellement aux muscles de la face, mais pouvant aussi en affecter d'autres, ceux du cou, du tronc et des membres . . . . Chez l'un, c'est un clignotement des paupières, un tiraillement convulsif de la jove, de l'aile du nez, de la commissure des lèvres, qui donne au visage un air grimaçant; chez un autre, c'est un hochement de tête, une contorsion brusque et passagère du cou se répétant à chaque instant; chez un troisième, c'est un soulèvement d'épaule, une agitation convulsive des muscles abdominaux ou du diaphragme; c'est, en un mot, une variété infinie de mouvements bizarres qui échappent a tòute description . . . . Ces tics sont on quelques cas accompagnés d'un cri, d'un éclat de voix, plus ou moins bruyant, très caractéristique . . . . Ce cri, ce jappement, cet éclat de voix, véritables chorées laryngées ou diaphragmatiques, peuvent constituer tout le tic . . . . C'est encore une tendance singulière à répéter toujours le même mot, la même exclamation; et même l'individu profère à haute voix des mots qu'il voudrait bien retenir . . . ."

No better way of introducing the subject and of giving the reader a good general idea of the disease under notice could possibly have been chosen than that adopted by the authors. They have devoted the first forty pages to "Les Confidences d'un Tiqueur," the latter being a gentleman who not only proves to be a perfect clinical museum of tic phenomena, but displays exceptional powers of introspective analysis, with the result that both the motor and the mental factors of the malady are exposed in a novel and instructive manner. Later on both these factors are discussed in further detail, and much emphasis is laid on the fact that in a true tiqueur there is always some mental as well as some motor disturbance; in other words the tic is the outward and visible manifestation of some psychical instability.

No light is thrown on the pathological anatomy of the malady, and the authors express their opinion that autopsies will always prove negative. "Nous ne croyons pas en effet que l'affection soit sous la dépendance d'une lésion acquise; nous supposons plus volontiers qu'il s'agit d'anomalies congénitales, d'arrêts ou de

vices développement des voies d'association corticales ou des anastomoses infracorticales, minuscules malformations tératologiques que nos connaissances anatomiques sont malheureusement encore impuissantes à réconnaître."

All the various forms of movement exhibited by the victims of this morbid tendency are discussed and illustrated by many clinical examples; the diagnosis and prognosis are dealt with in detail, and the last part of the book is devoted to the important subject of their treatment. The inconstancy of results obtained by the use of drugs, of baths, of electricity and of operative measures are well-known, and it would seem that the chief object of the writers is to advocate the method of re-education which, in their hands, has certainly been wonderfully successful. Relying on the fact that in all true tiqueurs there is, in the first instance, some deficiency in mental or moral power, they endeavour to supply this want by encouraging gradual systematic exercises which aim at developing self-discipline in the same way as the exercise of a muscle will develop the force of its contraction. The numerous methods and ingenious devices they have employed are given in full, and cannot fail to prove extremely useful to all who wish to follow in their footsteps.

E. Farquhar Buzzard.

PROCEEDINGS OF THE NEUROLOGICAL SOCIETY OF THE UNITED KINGDOM.

At the Clinical Meeting held at 11, Chandos Street, on Thursday, October 22, at 8.30 p.m. (patients in the room at 8 p.m.) the following cases were shown :—

(1) Dr. Wilfred Harris and Mr. V. Warren Low.—*Case of Erb-Duchenne paralysis, treated by operation.*

A man, aged 40, three years ago fell out of his milk-cart on to his left shoulder, causing immediate paralysis of his left arm, followed by wasting of the shoulder. He was treated by galvanism and massage at two well-known London hospitals, but no improvement resulted. In June of this year his condition showed complete paralysis and wasting of the left deltoid, supra- and infraspinatus, biceps, brachialis anticus, supinator longus, both radial extensors of the wrist, and the pronator radii teres. No reactions of these muscles could be obtained either to faradism or to galvanism. In addition there was distinct partial wasting and weakness of the whole of the left pectoralis major, and of the latissimus. There was very slight tactile anæsthesia on the outside of the left arm above the elbow, and definite, marked anæsthesia and numbness of the left thumb and radial side of the forefinger causing clumsiness of the hand, which he is quite certain dated only from the time of the fall.

Operation exposing the upper roots of the brachial plexus showed the fifth and sixth roots to be buried in scar tissue, the uppermost portion of the fifth root appearing to end in a swollen mass of scar tissue near the foramen, as though the root were partially ruptured. The scar tissue around both roots was dissected away as far as possible, and the fifth root cut across one-third of an inch above its junction with the sixth root, and its distal end sutured into a nick made in the upper border of the sixth root. Faradic stimulation of the sixth root gave moderate contractions of the pectorial, latissimus, triceps, and extensor

carpi ulnaris, with inward rotation of the shoulder, and slight contraction in the flexors of the wrist. Stimulation similarly of the fifth root evoked only slight fibrillary contraction in the wasted deltoid. Since the operation three months ago he has recovered perfect sensation on the thumb. No motor improvement yet.

(2) Dr. Wilfred Harris and Mr. V. Warren Low. *Case of infantile paralysis of the shoulder treated by operation.*

A girl, aged 2 years in July of this year, was attacked last February by infantile paralysis, causing paralysis of the right shoulder, which has not been recovered from. When seen in May, there was marked atrophy of the right deltoid, supra- and infraspinatus, with inability to raise the arm. The biceps and supinator longus were slightly weak, but she could flex the forearm. Electrical reactions taken under an anæsthetic showed complete reaction of degeneration in the deltoid and infraspinatus, but fair reaction to faradism in the biceps and spinator longus. Treatment for three weeks with galvanism made no difference to the condition, and it was quite clear that the shoulder would remain permanently paralysed unless something further could be done. Operation on the upper roots of the brachial plexus was performed, the fifth root being exposed and stimulated faradically, with resulting powerful contractions of the biceps and clavicular pectoral. The fifth root was then split longitudinally into two halves, upper and lower, and the two halves were then separately stimulated by faradism. With the upper half, only fibrillary contraction of the deltoid resulted, but stimulation of the lower half gave just as powerful contractions of the biceps as when the whole root was stimulated. It was, therefore, safe to divide the upper half of the fifth root, which was accordingly done, about one inch above its junction with the sixth root, and the distal end was turned down and sutured into a nick made in the upper border of the sixth root. Recovery from the operation was uneventful, with no further weakening of the biceps. It is still too early to expect any motor improvement in the paralysed deltoid.

(3) Dr. T. Grainger Stewart.—*A case of diplegia and myoclonus.*

W. D., aged 7, is the eighth child of a family of twelve. The parents are both healthy and there is no history of consanguinity, syphilis or nervous disorder. All the children are backward and

poorly developed, and two elder brothers were in hospital suffering from a similar complaint. The patient was always weakly and had several fits between the ages of 3 and 5. He could walk when 2 years old but has never spoken although he appears to notice what goes on around him. His present trouble came on with unsteadiness in the legs a year ago and he is now unable to walk. He is always happy and never cries. Rarely athetoid movements have been seen, but the most striking feature is the presence of sharp instantaneous shock-like contractions of various muscles, often of an individual muscle or part of a muscle. The contractions are seen in the muscles of the face, tongue, extremities and trunk; frequently corresponding muscles are affected simultaneously. These movements are not apparently felt by the patient or do not interfere with his grasping an object held out to him; they persist during sleep, but are less frequent. There is no evidence of congenital syphilis. The deep reflexes are brisk and the plantar reflexes extensor in type. His head suggests commencing hydrocephalus.

(4) Dr. Leonard Williams.—*A case of infantile hemiplegia of the right side, with paralysis agitans beginning on the left side.*

A woman, aged 39, who has the evidences of an infantile hemiplegia on the right side, and unmistakeable paralysis agitans on the left, states that she was born with her right side paralysed, a statement which is supported by her mother. The left-sided condition is attributed to a shock which she had in connection with her brother, who was a soldier in the late war. The movements on the right side have become less marked since the tremors on the left have developed.

(5) Sir Victor Horsley.—*Three cases of cerebellar tumour after operation, with reference to the question of cerebellar compensation.*

Abstract of Three Cases of Cerebellar Tumour Removed by Operation, illustrating Recovery of Function of Cerebellum.

Case 1.—T. S., aged 5, admitted into University College Hospital, July 18, 1902.

History.—Pain in the neck since October, 1901, loss of power in the legs May, 1902. May, June, and July, attacks of intense pain referred to the back of the neck followed by loss of consciousness. Fits of loss of consciousness together with moderate opisthotonos and extensor rigidity of arms and legs, double optic

neuritis, right more than left, slight paresis of right limbs; vomiting. The child was unable to stand without a great deal of support. Diagnosis was tumour of the right lateral lobe. Operation in two stages. At first stage dura opened on account of tension. Dark red tumour exposed. At second stage tumour found to be prolapsing through the opening in the dura. Considerable loss of substance of softened cerebellum. Microscopically tumour found to be glioma. Slow, but steady return of coördination, but cerebellar attitude of trunk and lower limbs present six months after operation. Child completely recovered one year after operation. All incoördination disappeared.

Case 2.—S. C., aged 7, admitted National Hospital for Paralysis, &c., April 7, 1903.

History.—Rapid onset, three week's headache, loss of sight, vomiting, incoördination, strabismus. Double optic neuritis, left more than right. Paresis, left sixth nerve. Cerebellar attitude, head to the left, head and chin turned to the left. Left occipital fossa more prominent than the right. On opening the dura the cerebellar tissue protruded and the folia were flattened. During the first twenty-four hours a large amount of fluid escaped from the cyst. Within eight days symptoms greatly diminished, and in one month all incoördination had gone.

Case 3.—A. W., aged 15.

History.—Gradual onset (4 months), severe occipital headache, vomiting, and difficulty in walking. Incoördination of left arm more than right. Double optic neuritis. Paresis, left arm. After first stage of operation considerable shock. At the second stage folia were flattened, a large cyst opened in the lobe containing brown fluid. Cyst drained. Gradual disappearance of incoördination. Patient completely recovered.

(6) Dr. DUNDAS GRANT.—*Three cases of nervous lesions with manifestations in the throat and ear.*

Case of Paralysis of the Palate and Larynx.

J. W., aged 25, has well-marked paralysis of the palate and of the abductors and internal tensors of the vocal cords. There is scarcely any separation of the arytenoid cartilages during inspiration, but there is between the cords an elliptical slit allowing of a fair amount of respiration. The voice is accordingly husky. The movements of the lips are somewhat clumsy and there is inability to channel the tongue. The protrusion of the

tongue into the right cheek has been observed to be not quite so strong as into the left.

The patient was first seen in March, 1900, on account of cough and a groaning sound when in bed. This condition lasted for two or three months, and it came on after an attack of hiccough lasting on and off for about ten days. For six months he had had occasional stridor on inspiration. At that time there were found enlarged post-cervical glands of about six months' duration, and in the right parietal region of the scalp a flat ulcer with slightly indurated edges, which was asserted to have been in existence for nearly three or four months. There was also general enlargement of the lymphatic glands over the body.

An anti-specific course of treatment was instituted, and when he was seen a fortnight later it was reported that there was less noise in sleep since the second inunction of mercurial ointment. The patient was not again seen by the exhibitor until 1902, his symptoms having in the meantime so much diminished that he was able to carry on his work as a butcher. He then again complained of difficulty and stridor in breathing, also of regurgitation of fluids through the nose, his speech being at the same time so indistinct that he was obliged to pinch his nostrils in order to make himself understood. He was again placed on antispecific treatment and his breathing and speech underwent slight improvement even within a week; he, however, drew attention to the fact that he had a difficulty in raising his left arm, apparently on account of a paralysis of the serratus magnus muscle, which, however, gradually disappeared.

The exhibitor is disposed to think that the ulcer originally present on the right side of the scalp was in some way the site of a primary specific infection, the improvement taking place under mercury and iodide of potassium apparently confirming this view.

Case of Hemianæsthesia affecting the Skin and Special Senses with Crossed Amblyopia and Achromatopsia.

Miss —, aged 41, a schoolmistress, frequently suffered from complete loss of voice and hoarseness since December, 1890. The voice had been restored from time to time by faradism and cauterisation of the throat. She has suffered from purulent rhinitis, affecting various sinuses, now mainly the sphenoidal, with frontal, postocular and occipital headaches. In 1895 it was discovered that she had hemianæsthesia of the right side of the face and body, including the olfactory, gustatory and

auditory nerves, with diminished visual acuity on the left side. The dimness of vision in the left eye is accompanied by inability to distinguish green and red from each other. The tuning-fork tests show the loss of hearing on the right side to be of the nature of nerve deafness, and it is as pronounced for the lowest as for the highest tones. Galton's whistle being heard up to the mark 3, practically the same as on the other side. The case is presumably functional.

Case of Paralysis of Vocal Cord with Signs of Involvement of the Sympathetic Nerve.

The patient, a married woman, aged about 40, was first seen in March, 1903 complaining of "discomfort" in her throat, which had come on suddenly three weeks before.

There is marked palatal paresis affecting pronunciation, and there has been regurgitation of fluids through the nose. The vocal cords perform only very limited excursions during inspiration, the left one hardly moving at all. During phonation the edge of the left vocal cord is concave, and it is so lax that it is blown upwards by the expiratory blast. The left palpebral fissure is narrower than the right, and there is persistent compensatory elevation of the left eyebrow. The left pupil is smaller than the right; it does not contract to light or on accommodation, and it dilates only very slightly under euphthalmine. The left side of the face perspires more than the right.

The exhibitor is desirous of eliciting an opinion as to whether the combination of symptoms is produced by a single or by multiple lesions. In view of the probability of the latter, he has looked upon the case as being of specific origin, and has found improvement to follow antispecific treatment.

(7) Dr. Gordon Holmes.—*Three cases of unilateral tremor in cerebral lesions.*

A. B., aged 17. A case of slowly progressive left-sided hemiplegia with irregular movements of the left limbs and inactivity of the pupils to light stimulation. A history of headache and vomiting, but no optic neuritis.

Diagnosis.—Thalamic tumour on right side.

R. E., aged 59. Six months ago sudden onset of weakness of the left limbs and inability to walk, no loss of consciousness. On admission to the National Hospital, four months later, there was complete paralysis of upward movement of the eyes, some limitation of downward movement, but lateral deviation and conver-

gence were normal. The pupils were inactive to light. There was a slight degree of weakness and rigidity of the left limbs, with an extensor plantar response on the left. Also coarse, but rather regular tremor of the left upper extremity in movement, and even during rest when excited or when the right limb was in action. Her gait was shuffling and stiff and face fixed and expressionless. There was no sensory change. The left-sided weakness is now much less and Babinski's sign has disappeared, but the tremor is more pronounced.

Diagnosis.—Vascular lesion of mid-brain and thalamus, chiefly affecting the right side.

C. S., aged 37. Slow onset of "uselessness" of the right limbs of two years' duration. Arm was affected for some months before there was any affection of gait. The trouble has slowly increased. There is a history of headache and vomiting but no changes in the fundi. There is no definite weakness of the right limbs as compared with the left, but she cannot use the arm owing to "unsteadiness." Coarse tremor of the right arm even when at voluntary rest, especially on excitement. There is some blunting of all forms of sensation on the right side of the body with marked loss of the sense of position. The reflexes are all normal and both plantar responses of the flexor type.

There is besides diplopia due to weakness of both externa recti, with nystagmus on lateral deviation. The pupillary reactions are normal. There is no hemianopia.

The tremor has diminished under antispecific treatment.

Diagnosis.—(?) Thalmic and mid-brain tumour.

(8) Dr. COLLIER.—*A case of* (?) *disseminate sclerosis.*

A man, aged 20 years. No antecedent illness. No lues. Symptoms of eight months' duration and progressive. Optic atrophy. Irregular ophthalmoplegia. Speech slightly altered. Jerky tremor of arms. Knee jerk absent. No ataxy of legs. No sphincter trouble. No pain. No anæsthesia.

(9) Dr. F. E. BATTEN.—*Case of* (?).

G. T., aged 4 years, second of three children; others well. Born at full term, no difficulty with labour. Began to walk when 18 months old but has never walked properly; has difficulty in getting up from the floor. Learnt to talk early, is clean in her habits and uses arms well.

A well-nourished child, intelligent; talks well, but is very slow in swallowing her food. All movements of face, eyes, lips,

and tongue well performed. Movements of the arms well and strongly performed, but there is very slight intention tremor.

Gait.—Stands with legs rather far apart but is perfectly steady, and when walking still keeps wide base. *Legs.*—Cannot kick out the legs well, and has difficulty in flexing them at the thighs. The foot cannot be dorsi flexed beyond a right angle owing to contraction of the gastrocnemius. Slight pes cavis. The knee jerks are absent, there is no ankle clonus. Both plantar reflexes give extensor responses. All muscles react fairly readily to faradism.

The condition is not progressive, and there is a tendency towards improvement.

(10) Dr. FARQUHAR BUZZARD.—*Progressive muscular atrophy in a young woman.*

L. S., age 28, housemaid, single. Between two and three years ago noticed gradual wasting of muscles of left hand. This has spread to some of the left forearm muscles, to the right hand, and to the muscles of the left foot and thigh. Loss of power and of electrical excitability in the muscles, involved proportionate to the degree of atrophy. No sensory changes. No affection of cranial nerves. No spinal curvature. Knee jerks active. No other members of the family affected.

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EDITOR.